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# INTRAVENOUS ADMINISTRATION OF ARSPHENAMINE IN THE TREATMENT OF ADVANCED FUSOSPIROCHETAL DISEASE OF THE ORAL CAVITY AND CONTIGUOUS TISSUES[1]

MALCOLM WALLACE CARR, D.D.S., F.A.C.D.

*Department of Oral Surgery, Metropolitan Hospital, Department of Hospitals, City of New York*

Experience indicates that the simple or moderately severe case of Vincent's stomatitis will respond reasonably well to the generally accepted methods of local treatment. Fusospirochetal infection, however, occasionally invades adjacent or contiguous soft tissues, causing sloughing of the deeper tissues over a considerable area.

Fusospirochetal disease is no longer considered solely a disease of the mouth. Smith[2] discusses in detail fusospirochetal infections found throughout the tissues of the body. The importance of fusospirochetes in certain pulmonary conditions was recognized some time ago, and now certain cases of pulmonary gangrene, pulmonary abscess, and bronchiectasis are believed to be due directly to fusospirochetal infection. The fusospirochetes found in pulmonary infections are believed to be the same type as found in the mouth, reaching the lung probably by direct aspiration.

Many clinicians have demonstrated the effectiveness of the use of arsphenamine intravenously in the treatment of bronchiectasis, pulmonary gangrene and pulmonary abscess, particularly in those cases where the fusospirochetes have been demonstrated to be etiological (or at least aggravating) factors. It is therefore logical to assume that intravenous use of arsphenamine is rational *in certain advanced cases* of fusospirochetal infection of the mouth, and also of adjacent or remote tissues where these anaerobes *cannot* be reached effectively by

[1]Presented at the Thirteenth General Meeting of the International Association for Dental Research, Chicago, March 16-17, 1935. *J. Den. Res.*, 15, 212 (1935).

[2]Smith, David T.: Oral Spirochetes and Related Organisms in Fuso-spirochetal Disease, Williams & Wilkins, Balto., 1932.

superficial local treatment. While experience demonstrates that there are definite indications for the use of arsphenamine in these types of cases, it is not recommended that it be used as routine treatment for Vincent's stomatitis. Since arsphenamine is a benzol derivative and as such is capable of depressing bone marrow, the drug should not be employed without some knowledge of the general condition of the patient, for example, the differential blood picture. Secondary anemia, leukopenia and agranulocytosis, as well as albuminuria, may contraindicate its use.

Arsphenamine employed intravenously is usually best given in small doses [300 mgm. (0.3 gm.) in 30 c.c. normal saline], once a week for three or four weeks, or the initial administration may be repeated within three or four days in acute cases. Usually one or two administrations suffice. In pulmonary infections potassium iodide is also given, with proper attention to rest and diet; all of which is applicable where arsphenamine is being used to treat mouth infections. In the case of mouth infections abstinence from smoking is an important factor. There is no reason why local treatment may not *also* be carried on.

The following case report illustrates the effective therapeutic value of the use of intravenous arsphenamine:

G. O.: Male 68 years of age. Admitted to hospital with extensive cervico-facial cellulitis, and submasseteric abscess from infected maxillary molar teeth.

*Bacteriological examination:* Smear from gingival tissues positive for fusospirochetes. Smear from throat negative.

Culture from neck wound: Aerobic streptotoccus hemolyticus, staphylococcus hemolyticus aureus, anaerobic streptotoccus hemolyticus.

Smear from neck wound: Predominating organism a gram positive diplococcus, present both outside and inside of pus cells. Vincent's spirochete and fusiform bacilli also present in great numbers.

*Hematological examination:* Leukocytes—14,600 per cu. mm.; Polymorphonuclears—86 per cent; Lymphocytes—12 per cent; Monocytes—2 per cent.

*Serological examination:* Wassermann and Kahn reactions negative.

*Urinalysis:* Negative.

*Treatment:* External incision 2 cm. beneath the lower border of the mandible, extending from the median line posteriorly to just anterior to

the angle. Dissection was done down through the platysma. Edema made it difficult to identify the structures throughout entire region. At posterior margin of incision a piece of necrotic, black, foul smelling fascia was found which seemed to be the deep cervical fascia of the neck, and which, when incised, did not enter an abscess cavity but released a small amount of foul-smelling, chocolate colored pus.

This was an exceedingly severe infection, patient was very septic and ran a high temperature (103°) for more than a week. Slough extended and two days after operation the patient was given intravenously 300 mgm. (0.3 gm.) of neoarsphenamine in 30 c.c. normal saline. This was repeated 48 hours later. Slough from the neck was removed two days later, and although there was slight drainage, wound was free of necrotic material. A slough from a wound of this nature ordinarily would not come away until about the tenth day. The neck incision was allowed to close in about one week. The intra-oral condition improved more slowly. The infection later ascended along the masseteric fascia and beneath the temporal fascia, and a secondary operation for drainage was done in the temporal region with counter drainage established intra-orally. The suppuration continued for a number of weeks but eventually the patient made a complete recovery.

The advantage of the supplemental use of arsenic preparations (arsphenamine and neoarsphenamine) in the field of oral surgery has been demonstrated in several conditions arising from fusospirochetal infection. These conditions may be classified as follows: (a) Advanced fusospirochetal stomatitis with deep ulcerations in adjacent mucous membrane surfaces; the buccal mucosa, soft palate, floor of the mouth, the tongue and the lips. (b) Advanced fusospirochetal infection of the mouth concomitant with an acute secondary suppurative process (cellulitis) in which fusospirochetal organisms have been demonstrated at operation in the deep submaxillary contiguous tissues. (c) Fusospirochetal infection of the mouth (stomatitis) and the pharynx with secondary extension to remote tissues—the middle ear and mastoid process.

# THE CONTROVERSY CONCERNING THE INNERVATION OF THE TEETH[1]

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In studying the literature on the histology of the tooth, one is impressed with the want of unanimity in the views expressed by various writers. One cannot select a single tissue entering into the formation of the tooth, whose function is not, even today, interpreted in several ways. This is especially true in the problem of nerve fibers ending in association with the odontoblasts or penetrating the dentin and terminating in its substance. This is not due to the lack of careful work, but rather to the difficulty which surrounds these investigations.

To search for the first allusions to nerves in the teeth is to go back to antiquity. The time when dental pain was not discussed is beyond our knowledge. Galen (Taylor, 1922) in his anatomical researches recognized seven pairs of cranial nerves and classified the trigeminus as the third. Galen also records two sites of pain, one in the tooth and the other in the surrounding tissues (Latham, 1901). Aetius of Amida, 502–575, a celebrated Greek physician and author stated the cause of toothache was known only to God. He taught that the mucous membrane of the mouth tissues is provided with nerves from the 3rd pair of cerebral nerves and that the teeth, too, by a small hole existing at the end of every root, receive tiny ramifications of sensitive nerves, having the same origin (Weinberger, 1926).

Leeuwenhoeck (1678) gave the first description of the tubular nature of the dentin from observations made on one of his own teeth. However it was not until some 170 years later that the dentinal tubules were cited as being a possible structural element whose contents entered into the cause of dental pain.

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John Hunter, who was the first to describe accurately the growth of the jaws, (Brodie, 1934) recorded his opinion regarding dental pain in his Natural History of the Teeth (1778). "In the cavity of a tooth it is well known that there is exquisite sensibility; and it is likewise believed that this is owing to the nerve in that cavity. This nerve would seem to be more sensible than nerves are in common; as we do not observe the same violent effect from any other nerve in the body being exposed either by wound or sore, as we do from the exposure of the nerve of a tooth. Perhaps the reason for the intenseness, as well as the quickness of the sense of heat and cold in the teeth, may be owing to their communicating these to the nerve sooner than any other part of the body."

Duval (1833) showed that there is sensation in the dentin chiefly manifest just below the enamel (Mummery, 1911–12).

In 1835, Raschkow asserted that the pulp has one large and a few smaller nerves, the latter breaking up near the surface of the pulp into an exceedingly fine plexus which he called the plexus of Raschkow (Latham, 1901). Owen (1840–45) stated that the nerves "penetrate the orifices of the fangs and form a rich plexus with rhomboidal meshes upon the coronal surfaces of the pulp; the peripheral elementary filaments returning into the plexus by loops." Further, Owen, citing Purkinje and Raschkow (1835) in regard to the formation of dentin, said these two observers noted that embryologically true nervous filaments cannot be distinguished in the pulp until its vascularity had been established.

Dental histology had its beginning about 1840. It was not until 1835 that the compound microscope was perfected and the minute organization of the dental tissues studied in detail.

John Tomes in 1848 described what he called nerve fibrils in the dentin continuous with the pulp network and considered these fibrils to be the cause of sensitive dentin (Latham, 1901). Later Tomes (1856) carefully and successfully continued the study of the finer point of dental structure by demonstrating these fibers as dentinal fibers (Tomes' fibers). In 1856, Tomes stated that "The recognition of the fibrils of the dentine will, however, I think, remove the difficulty, and enable the physiologist to explain why under certain circumstances that tissue is susceptible to pain, while under other conditions the

sensitiveness is lost. That the dentine owes its sensation to the presence of the dentinal fibril cannot, I think be readily doubted, seeing that if their connection with the pulp be cut off by destruction of the latter, all sensation is lost." Waldeyer (1871) speaking of Tomes stated "he opened the way to a correct interpretation of the nature of the dentine."

Probably the first to state the opinion that the nerve fiber endings are in the dentin was Beale (1860). He said that there is room in the dentinal tubules for whole plexuses of minute nerve fibrils, and there is reason to suppose that they enter the tubules in common with the germinal matter (odontoblasts and processes), the tubules measuring 1/10,000 of an inch while these minute filaments are but 1/100,000 of an inch.

Fitch (1864) held the idea that the dentin is endowed with nerve fibrillae, perhaps so minute as to elude microscopical investigation or perhaps "permeated with that which takes place of a nerve fiber, which position involves the thought that impressions are susceptible to transmission by other than nerve substance." Francis (1865) compared the sensation in the tooth with ghostly tenants inhabiting every cell of the dentin and like trusty sentinels giving warning of approaching danger. He further stated that this "dentinal life-presence" was not easily photographed and it must emanate from the membrane sac which invests the pulp, and this "life-presence" was probably an effusion of "liquor sanguinis" bearing the character and mission of nerves.

Some of the current theories of that period were that there were nerves and blood vessels in the dentinal tubules; that the dentin was charged with electricity, the electrical fluid communicating with the nerve which responded to outside influences; and that the dentine contained some subtle fluid, which when touched with an instrument was forced back upon the nerve eliciting pain.

McQuillen (1866–67) was of the opinion that the dentine owes its sensation to the presence of the dentinal fibers. He further stated that if nerve fibers were distributed to all the dentinal tubuli every tooth would be exquisitely sensitive not only to manipulations of the dentist but even sound teeth would ache when exposed to thermal changes of everyday life. Further the dentinal tubules tend to be-

come obliterated with advanced age and if nerve fibers were distributed to the tubuli this obliteration would cause excruciating pain and suffering. Burgh (1867) considered sensitive dentin a pathological phenomenon but gave no reason for this assumption. Cutler (1867) compared the pulp chamber of the tooth with the interior of the cranium and thought the dentinal tubuli were occupied with nerves like cranial nerves in the foramen emerging from the skull.

In this development of the study of the innervation of the teeth, improvements in the preparation of microscopic specimens have played a great part; thin hard sections and decalcification were apparently employed very early, and many refinements had been introduced such as grinding and freezing. "Early investigators had no suitable stains and the development of selective staining technic has done much to solve some of the most difficult problems of microscopic anatomy of the teeth; such for instance, as the innervation of the teeth" (Denton, 1934).

Up to 1868 no constructive work had been done toward demonstrating these observations other than that done by Beale and a few others, the histological work being more or less ineffective. Boll (1868) thought he could demonstrate fine nerve fibers in the dentin and hence put forth an histological basis for the theory that nerve fibers ended in the dentin. His work was done with pulps of animals' teeth stained with chromic acid and gold chloride. He was able to trace medullated and non-medullated fibers between the odontoblasts and though he was unable to see the nerve fibers enter the dentinal tubules he assumed such an entry as probable. Waldeyer (1871), commenting on the work of Boll, stated "We owe to Mr. Boll (a medical student at Bonn) the first definite knowledge of the condition of nerve fibers in the teeth."

White (1871) in England made the assumption that the processes of the odontoblasts become fused with the outer coat of the nerve while Coleman (1871) in the discussion that followed White's paper regarded the odontoblasts as comparable to the tactile corpuscles, Pacinian bodies, rods and cones of the retina and other special receptors. Harriman (1871) in Boston, claimed to have demonstrated by ground sections a small nerve fibril on several dentinal fibers and also to have been able to dissect out one of these nerve filaments, which were less

than 1/50,000 of an inch in diameter. He used acid to decalcify the tooth. Salter (1874) stated that nerves of the pulp formed loops toward the periphery of the pulp and it was highly improbable that they pierced the wall of the odontoblast and occupied the axis of the odontoblastic process.

Charles Tomes, son of John Tomes, put forth the explanation that the odontoblast is a specialized receptor comparable to the goblet cell in the olfactory membrane of the frog. Legros and Magitot, founders of our modern conception of the embryology of the teeth, examined the pulps of teeth of animals with gold chloride and observed the nerve fibers of the pulp to be directly continuous with the central processes of the odontoblasts. Klein (1883) stated that the medullated fibers of the pulp lose their medullary sheath toward the periphery of the pulp and the axis cylinders probably ascended in the dentinal tubuli. C. F. W. Bödecker (1882), also using gold chloride, observed the same processes of the nerve fibers losing their myelin sheaths but he found the axis cylinders ended in knob-like extremities or branched between the odontoblasts and he positively asserted these nerve fibers made an indirect connection with the odontoblasts. Sudduth (1886) agreed with Bödecker but further stated that the terminal fibers united with the odontoblasts. Black (1887) was of the opinion that the dentin derived its sensory function direct from the pulp through the dentinal fibers.

Robertson (1890) said the odontoblasts in the teeth of oxen were bipolar. Later (1891) he asserted that the odontoblasts are neurosensory cells whose basal processes are directly continuous with the nerve fibers of the dental pulp. Morgenstern (1892) using the Golgi method and methylene blue described nerve fibers passing into special canals in the dentin and ending in ellipsoidal bodies. Rose (1893) who saw Morgenstern's preparations, repeated his work and reached the conclusion that Morgenstern's nerve endings were only areas of uncalcified ground substance through which the dentinal fibers pass.

Hopewell-Smith, the leading advocate of the school that believed nerve fibers terminated in the odontoblast layer, (1893) considered that the odontoblasts are solely for the transmission of nervous impulses but was not able to establish any connection between the nerve fiber and the odontoblast. Retzius (1892–93–94) described the course

of nerves of the pulp of teeth of different classes of vertebrates. Using the Golgi method he observed the nerve endings as free, beaded terminations of fine fibers among the odontoblasts and between the odontoblasts and the dentin. Hoehl (1896), from the results of his work, held a similar view. Stack (1896) tried to show that there is a special development of nerve endings reaching as far as the internal surface of the enamel, basing his opinion on the very delicate sense of touch possessed by the tooth.

In 1898 Huber, using the intra-vitam methylene blue method on animals found that nerves as they entered the pulp were of one or several relatively large bundles of medullated fibers. They divided into numerous smaller bundles and formed medullated plexuses. On approaching the periphery of the pulp they lost their medullary sheath, formed small plexuses underneath the odontoblasts and had free beaded endings near the free end of the odontoblasts. Burchard (1898) made similar observations but doubted the relationship of the nerve fiber to the odontoblast. Romer (1899) claimed to have demonstrated that unmyelinated fibers passed into the interior of the odontoblast process and ran with these into the dentinal tubuli.

Gysi (1900), reviving one of the earlier theories, stated that the free nerve endings are stimulated by pressure on the fluid content of the dentinal tubules. Pont (1900), evolving a somewhat fantastic theory, asserted that the odontoblasts resemble peripheral sensory neurons and are, therefore, in reality, cells of the nervous system, the dentinal processes of which are the centripetal poles and the basal processes the centrifugal poles. Latham (1902), attempting to explain the termination of nerves in teeth, said she had seen no work stating or even demonstrating the odontoblast as mesoblastic in origin and further that the odontoblast might be epiblastic in origin. She compared the ending of these nerves upon the odontoblasts with the well established termination of nerve fibers upon gustatory cells and similar sensory cells. To support this concept she demonstrated, nerve fibers in the odontoblastic plexus and occasionally traced one into the dentin.

Hopewell-Smith (1903) was able to show that the nerve fibers of the pulp, "terminate in a basket work of varicose fibers, embracing and often closely attached to the cell wall of the individual odontoblast."

Law (1907–08) using the Bethe and Golgi methods claimed that he was not able to obtain any appearance of nerve fibers actually entering the odontoblast cell. He believed that the nerve fibers run direct to their destination and the supposed plexus of Raschkow was merely made up of bundles of connective tissue fibers. Fischer (1909) decided that the nerve fibers end in association with the odontoblast.

Mummery, the leading British advocate of the view that the dentin contains nerve fibers, started his work as far back as 1891 but it was not until 1910 that he was able to satisfy himself that nerve fibers enter the dentin. In a series of papers from 1910 to 1913 he stated his opinion, based on work with Beckwith's gold chloride method, that the nerve fibers form a marginal plexus and enter the dentinal tubuli in company with the dentinal fiber. He claimed he could trace the nerves as fine beaded fibers terminating in the fine ramifications of the dentinal tubuli beneath the enamel and cementum. Dependorf (1913–15) concluded that the dentin is innervated with two types of nerve fibers, one in the dentinal tubules and the other in the ground substance of the dentin. Fritsch (1914) using the Bielschowsky method, believed he could confirm some of the observations of Dependorf. At this time (1916), Mummery had some doubt of the existence of the marginal plexus, mentioned before, and decided that nearly all of the nerve fibrils passed directly into the dentin.

Evidence of the controversy regarding the problem of the innervation of the teeth appears in the correspondence and papers of Mummery and Hopewell-Smith. Hopewell-Smith (1916) stated that Mummery's nerve fibers in the dentin were connective tissue fibers. He stated further that nerves "terminate similarly to the telondria of the other ordinary sensory nerves, in exceedingly minute branchings about the odontoblasts on the surface of the pulp and do not penetrate the dentinal tubules. This is my opinion and can be demonstrated by suitable methods of staining."

In defense of his work Mummery said, "I am glad I have at last obtained a definite statement from the author (Hopewell-Smith) as to the nature of these fibers I have figured and described. He says they are connective tissue fibers." The following letters were offered by Mummery as evidence that his fibers in the dentin were of a nervous origin. A letter from Sharpey-Schafer to Mummery stated,

"I am quite convinced of the nervous nature of the wavy fibrils. Dentin seems to be more richly supplied with nerves than any other tissue in the body." In a later letter Sharpey-Schafer said, "I have no doubt whatever that the fibrils you describe and figure are nervous —you are quite at liberty to say so." Starling wrote Mummery, "If the fibrils I saw in your specimens were not nerve fibers, I have not seen nerve fibers." Charles Tomes' letter to Mummery disclosed, "The onus of proof appears to me, therefore, shifted to showing that they are not nerves, and to demonstrating that they are something else, which seems to me to be very unlikely." From Underwood, Mummery quoted, "I have not seen only one, or two, but a great many of your specimens during the last few years, and I have repeatedly followed nerve filaments right into the dentine. . . . I have no lingering doubt whatever, and the established fact that nerve filaments pass into the substance of the dentine must now pass into current teaching throughout the scientific world."

In 1919–20 Mummery found structures interposed in the course of the nerves, which he considered as "nerve-end cells" placed in a row at the bases of the odontoblast cells. Mummery called the distal process of these cells entering the dentin the axon and the peripheral process surrounding the odontoblast, and having synaptic relations with the plexus of Raschkow, the dendrites. Hopewell-Smith (1919) said, "As a result of his researches the author (Hopewell-Smith) has the strongest conviction that these fibers terminate in a basketwork of varicose fibers embracing and often closely attached to the cell wall of the individual odontoblast."

Hopewell-Smith (1924) continuing this controversy reviewed extensive evidence to indicate the non-innervation of dentin and stated in conclusion: "There are no nerves in the dentine: the animal economy does not require them in this or any other calcified connective tissue. There is, consequently, no sensation in the dentine. There is no hypersensitivity of dentine." On the other hand Mummery gave evidence based on his own work and the work of others and said, "I hope I have proved to your satisfaction that the dentine is supplied with nerves. I feel that further research is necessary with regard to the cells (nerve-end cells), but the main fact of the innervation of dentine is, I hope, fully established."

Montfort as early as 1914 claimed to have found nerve end cells within the dental pulp. In 1920 and 1923 the observations of Montfort were similar to those of Mummery, but this investigator added a third element, "the nerve end cells of the central pulp," to the already complex picture. He also stated he noticed the existence of neuroblasts and embryonic nervous cells in dental follicles of newly born cats. Stewart (1927) undertook to repeat Mummery's work using the latter's method. He was able to demonstrate the fibers of Mummery running into the dentin. He showed that section of the inferior dental nerve did not cause these fibers to degenerate. He could not demonstrate the nerve end cells of Mummery or Montfort. Later Stewart (1929) found that the fibers in the dentin were present after ligation of the trigeminal and sympathetic nerve supply.

Applying electro-osmosis to specimens to remove excess acid after decalcification, Tojada (1927) and also Dieck (1927) found nerve fibers entered the dentinal canals where they accompanied and lay upon the dentinal fibers. The nerve fibers were traced from one-half to two-thirds way into the dentin. Downs and Goss (1929) by means of pyridine fixation, Cajal technic of silver impregnation and careful grinding of teeth claimed to show incontrovertible evidence that the dentinal tubules contained definite unmyelinated nerve fibers. Chase (1929), using eleven differential staining or impregnation methods showed that the fibers described by Mummery and others as nerve fibers were not such, but were V. Korff's dentinogenic fibers. The nerve end cells described by several were not neurocytes, but fibroblasts concerned in the formation of V. Korff's fibers. Chase further found that the nerve fibers in the odontoblast layer were arranged as described by Retzius, Huber and others. Cabrini (1928) traced a similar course for nerve fibers but could not decide whether they entered the dentinal tubules or not. Papa in 1929 (Windle, 1934) described free endings for nerve fibers among and between the odontoblasts and dentin.

Noyes (1921) and Orban (1928), have taken opposite views on the question. Noyes referred to Mummery's work and said, "Support for almost any idea can be found in the literature but many of the conditions described have been shown to be errors in microscopic interpretations." Referring to the work of Huber (1898) he stated further,

"The nerve fibers have never been followed into the dentinal tubes," and thus adopted the theory of conductivity of sensation by the dentinal fibril and odontoblast. Orban on the other hand has stated: "The nerve fibers can be traced not only to the dentin, but into the dentin itself, as has been shown by Mummery, Morgenstern, and recently by Adrion (1926) and Toyada-Dieck (1927)."

Calderon (1931) using the Cajal technic on the teeth of rats reached the conclusion that nerve fibers from the sub-odontoblastic plexus (Raschkow) ramified among the odontoblasts ending in expansions which are close to the nuclei of these cells. No nerve fibers were seen to enter the dentinal canaliculi or matrix. Tiegs (1932) using the Bielschowski method stated that the nerve fibers ended as fine varicose branching filaments that run for considerable distances underneath the dentin and terminated on the sub-dentinal odontoblastic processes by definite end organs. Some very delicate nerve fibers appear to terminate by minute swellings (loops) on the bodies of the odontoblasts. No nerve fibers were observed to enter the dentin and no nerve end cells were seen in the pulp.

Recent foreign works bring to our attention the still existing controversy regarding the termination of these nerve fibers. Gordon and Jorg (1933) described the nerve fibers as terminating in spherical beads in the "pre-dentin" layer and stated that as far as they were able to determine, the fibers do not have any contact with the odontoblast or its process. Riegele (1934) described an innervation of dentin by means of fibers, which after a short or long course in the dentine, alter their direction and appear to form a terminal nerve net. The neurofibrils which he observed within the dentin are said to be surrounded by nucleated neuroplasma sheaths.

When these more modern views have been studied it will be found that they fall into two distinct groups, the members of the first believing that a continuity exists between the nerve fibers of the tooth pulp and the odontoblasts and its process, the members of the second group claiming to have traced the nerve fibers into the dentin.

Developments in experimental methods of neurological research make it necessary to turn to the present conception concerning the mechanism of reception and conduction of pain from the pulp and dentin. As early as 1910 Spitzer and later Perna (1913) and Allen

(1925) removed the teeth of animals to observe the type and sizes of nerve cells in the semilunar ganglion undergoing chromatolysis. Windle (1927) extracted pulps of teeth in a series of animals and histologically examined these pulps and the gasserian ganglia after time allowed for axone reaction. In the ganglia he found that small cells predominately underwent chromatolysis. The fibers of the pulp were largely of the small myelinated variety, and they ended in the odontoblast layers as free, beaded terminations. Williams (1898) had emphasized the rôle of the vasomotor nerves of the pulp and showed that non-medullated branches of the pulp were distributed to the blood vessels.

At the present time we find that the anatomic basis for pain conduction is receiving active attention. Pain conduction in the trigeminal nerve and in other parts of the body has been shown to be conducted by the small nerve fibers, both myelinated and unmyelinated [Windle (1926), Ranson (1931), Ranson and Davenport (1931), and Davenport and Ranson (1931), and others]. Heinbecker, Bishop and O'Leary (1933) have presented evidence that the unmyelinated fibers take no demonstrable part and that the small myelinated fibers are predominately concerned in the phenomena of pain conduction. Adrian (1928) expressed the current opinion of neurologists that there are special receptors and conductors for pain and the evidence at hand indicates that free nerve endings in the corium, in the cornea of the eye and free endings elsewhere are responsible for pain reception. It is unnecessary for them to reach the surface of the body; they are stimulated through a medium of other non-nervous elements. Stopford (1930) has stated, "In the case of pain spots, histology has tended to suggest that there are no special receptors, and that the termination is by free endings of the fibers."

It is common knowledge that pain is the only sensation elicited by stimulations of nerve fibers which course within the pulp of the tooth. The present trend is to correlate reception of painful impulses in the teeth with the reception of like impulses in the rest of the body. This, in the case of the tooth pulp, indicates that painful stimuli are received by free nerve endings through the medium of the odontoblast and its process.

In spite of the earnest labors of over one half century, it is amazing

that while so much is known about the nervous system of the dental pulp, much is still unknown—the actual methods of the peripheral distribution of the nerves being buried in obscurity. True, that some of the earlier histologists solved most carefully *in suis modis* this particular and difficult problem; true, that modern methods of preparing the hard and soft tissues for microscopical examination have shed new light on it; it still remains that the direct course by which sensation is conducted from the dentin to the cortical areas of the cerebral hemispheres has not been satisfactorily determined.

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# STUDIES ON THE CHEMISTRY OF MIXED HUMAN SALIVA

## II. Sodium, Potassium and Calcium in Salivas Secreted at Widely Varying Rates[1]

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In a preceding report (1) three groups of salivas produced at widely varying rates were subjected to chemical analysis. It was shown that the quantity of ash, total solids and chlorides tended to increase while the phosphates tended to decrease as the rate of flow of the salivary secretion increased. In the present work,[2] carried out one year later, a similar study has been made in an attempt to correlate rate of secretion of saliva with its sodium, potassium and calcium content. All but three of the original group of eighteen donors were available and have contributed one hour specimens, under paraffin stimulation. To round out the group three more donors were selected, who produced at a rate which fell into the desired group rates. Details of collection were the same as reported previously.

### METHODS OF ANALYSIS

Analyses were made on specimens which had been centrifuged at the time of collection and which were thoroughly shaken before sampling.

Sodium and potassium determinations were made on a filtrate prepared as follows: Pipette 35 cc. of saliva into a flat 50 cc. platinum evaporating dish. Acidify with three drops of concentrated sulphuric acid and evaporate to dryness on a hot plate. Place the dish in the muffle furnace and ash at 650°. (The sulphuric acid is added here in order to convert the alkali metals to sulphates, which are not likely to volatilize during ashing.) Cool and add exactly 35 cc. of distilled water to the dish. Disintegrate and dissolve the ash by stirring with a small glass rod. Filter through an ashless paper.

[1] Presented at the Fourteenth General Meeting of the International Association for Dental Research, Louisville, Ky., March 14–15, 1936; *J. Den. Res.* 15: 320, 1936.

[2] We are indebted to the students who donated the saliva specimens and to the Columbus Dental Manufacturing Company for a grant to defray a part of the expense of this work.

Sodium determinations were made on two 5 cc. portions of this filtrate by the direct method of Cally and Foulk, (2) employing magnesium uranyl acetate as the precipitating agent. Alcohol was used as wash liquid, a correction of 1 mg. for every 5 cc. of wash liquid being added to the final weight of the precipitate.

Potassium was determined in 10 cc. portions of the filtrate by the perchlorate-butyl alcohol method of G. F. Smith (3).

Calcium was determined by the Clark-Collip modification of the Kramer-Tisdall Method, (4) on the original saliva specimen without deproteinization.

The analytical data on the eighteen salivas follow:

TABLE 1

*Résumé of mineral analyses of salivas*

| SUBJECT | VOLUME IN CC. | | | | | TOTAL SOLIDS | ASH | Na | K | Ca |
|---|---|---|---|---|---|---|---|---|---|---|
| | 1st 15 min. | 2nd 15 min. | 3rd 15 min. | 4th 15 min. | Total 1 hr. | % | % | mgm./ 100 cc. | mgm./ 100 cc. | mgm./ 100 cc. |
| "High-producing" Group | | | | | | | | | | |
| W. I. W. | 72 | 60 | 71 | 74 | 277 | 0.624 | 0.301 | 83.3 | 78.9 | 6.50 |
| C. B. R. | 82 | 64 | 65 | 64 | 275 | 0.722 | 0.326 | 131.1 | 93.0 | 5.13 |
| J. H. M. | 69 | 64 | 67 | 58 | 258 | 0.582 | 0.301 | 81.7 | 90.2 | 3.99 |
| R. F. D. | 77 | 61 | 47 | 54 | 239 | 0.412 | 0.174 | 23.3 | 93.3 | 6.61 |
| H. C. M. | 64 | 52 | 43 | 49 | 208 | 0.429 | 0.186 | 55.6 | 57.2 | 5.02 |
| R. L. S. | 49 | 39 | 50 | 65 | 203 | 0.559 | 0.255 | 88.3 | 75.5 | 3.99 |
| *Average* | **68.8** | **56.6** | **57.1** | **60.6** | **243.1** | **0.564** | **0.257** | **77.2** | **81.6** | **5.21** |
| "Medium-producing" Group | | | | | | | | | | |
| W. B. H. | 45 | 41 | 40 | 45 | 171 | 0.496 | 0.265 | 69.9 | 74.1 | 4.11 |
| H. J. C. | 42 | 36 | 42 | 36 | 156 | 0.717 | 0.355 | 118.2 | 71.0 | 5.70 |
| N. W. S. | 48 | 34 | 34 | 25 | 141 | 0.554 | 0.305 | 72.0 | 69.4 | 4.11 |
| F. E. W. | 35 | 30 | 35 | 33 | 132 | 0.443 | 0.246 | 65.3 | 76.1 | 3.42 |
| H. C. K. | 30 | 31 | 33 | 37 | 131 | 0.481 | 0.291 | 17.1 | 86.8 | 5.02 |
| P. D. | 41.5 | 28.5 | 36 | 34.5 | 130.5 | 0.420 | 0.130 | 86.4 | 115.9 | |
| *Average* | **40.2** | **33.3** | **36.6** | **35.1** | **143.3** | **0.518** | **0.265** | **61.5** | **85.5** | **4.47** |
| "Low-producing" Group | | | | | | | | | | |
| R. E. T. | 26 | 21 | 20 | 16 | 83 | 0.455 | 0.183 | 25.3 | 85.4 | 3.53 |
| J. G. | 26 | 17 | 18 | 16 | 77 | 0.583 | 0.196 | 40.0 | 91.3 | 4.11 |
| R. W. | 17 | 16 | 12 | 13 | 58 | 0.610 | 0.311 | 36.9 | 93.0 | 3.76 |
| T. W. S. | 16 | 17 | 12 | 12 | 57 | 0.460 | 0.189 | 16.5 | 75.0 | 4.79 |
| A. D. C. | 12 | 12 | 14 | 12 | 50 | 0.562 | 0.264 | 18.5 | 93.0 | 4.90 |
| D. W. | 13 | 12.0 | 11.5 | 11.5 | 48 | 0.440 | 0.197 | 29.4 | 75.3 | |
| *Average* | **18.3** | **15** | **14.5** | **13.3** | **61.1** | **0.518** | **0.223** | **27.7** | **87.1** | **4.21** |

## DISCUSSION OF RESULTS

The above results for ash are decidedly higher than those obtained in the previously reported series. This we believe to be due to the fact that we have employed in this work a pyrometer-controlled furnace, the temperature not being allowed to rise above 650°C.

The values for potassium, ranging from 57 to 115 mgm./100 cc., show decided individual variations, but the averages for the groups are very close, there being, if any variation, a slight tendency to decline with increase in the rate of secretion. Calcium values, on the other hand, appear to increase as more saliva is produced. The most variable mineral constituent of the three studied is sodium which is nearly three times as concentrated in the high producing group as in the low. For purposes of comparison the values of several blood serum constituents are shown along with those of the three saliva groups in the following table.

TABLE 2

| ELEMENT | SERUM[1] | LOW-PRODUCING | SALIVAS MEDIUM PRODUCING | HIGH PRODUCING | AVG. THREE GROUPS |
|---|---|---|---|---|---|
| | | *mgm./100 cc.* | | | |
| Sodium | 330 | 27.7 | 61.5 | 77.2 | 55.5 |
| Potassium | 19 | 85.5 | 85.5 | 81.6 | 84.5 |
| Calcium | 10 | 4.2 | 4.4 | 5.2 | 4.6 |
| Phosphorus[2] | 3–5 | 16.9 | 14.4 | 12.4 | 14.6 |
| Chloride[2] | 300 | 39.0 | 51.0 | 63.0 | 51.0 |

[1] Calculated from data in Hawk & Bergeim (5).

[2] These values for salivas taken from preceding report (1).

Serum contains more sodium, chlorine and calcium and less potassium and phosphorus than saliva. The high-producing salivas apparently tend to maintain their osmotic relationships at the expense of sodium chloride from the blood. These results with respect to sodium and potassium agree very closely with those of Gregersen and Ingalls (6) who determined these minerals in dog's submaxillary saliva resulting from chorda and pilocarpine stimulation.

## SUMMARY

In an analysis of three groups of six salivas each, secreted at widely varying rates, sodium increased rapidly and calcium slightly with

rate of secretion. Potassium, on the other hand, tended to decline slightly with increase in rate of secretion.

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# THE EFFECT OF SILVER NITRATE IN THE PREVENTION OF DENTAL CARIES

## I. The Effect of Silver Nitrate upon the Decalcification of Enamel[1]

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Clinical experience has indicated that the treatment of a tooth with silver nitrate will retard the destructive action of dental caries upon the enamel. The manner in which this retardation of decalcification is brought about is not clearly understood nor has it been thoroughly investigated. This paper deals with the influence of silver nitrate on tooth enamel when subjected to the action of weak organic acids. Other studies of the bacteriological aspects of the problem are in course. In spite of the many references to its clinical value and the investigations that have been made, an understanding of the manner in which this salt protects tooth structure against caries is not clear.

As early as 1846, dental periodicals suggested the advantage of the clinical use of silver nitrate in treatment of extensive dental caries. Miller (1) studied the therapeutic value of silver nitrate for the control of dental caries and the elimination of hypersensitive dentin. His attempt to show a retardation of decalcification of enamel when treated with silver nitrate was unsatisfactory, but he was able to demonstrate definite retardation of decalcification in similarly treated ivory.

Szabo (2) believed that the silver nitrate reacted with the organic material of the dentin, resulting in the formation of silver albuminate. Further protective reaction in the dentin was due, he thought, to a mechanical blocking of the tubules. Rickert (3) reported that teeth treated with silver nitrate became "clogged with reduced silver and

[1] Presented at the Fourteenth General Meeting of the International Association for Dental Research, Louisville, Ky., March 14-15, 1936; *J. Den. Res.*, 15: 308, 1936.

refused to act as a permeable membrane." He believed that enamel treated with silver nitrate was more resistant to the influence of weak organic acids and states, "This is not due alone to the approximate insolubility of silver but also to the impossibility of acids penetrating or diffusing through the tooth structure."

An attempt was made to reproduce the experiments of Miller using enamel instead of ivory. Teeth, of which one portion was protected with wax, were subjected to 5% and 10% solutions of silver nitrate for varying lengths of time. After treatment the wax was removed and the teeth thoroughly washed. These teeth were then treated with different dilutions of nitric acid for different lengths of time. When the teeth were removed from the acid, those surfaces treated with silver nitrate were not etched to as great an extent as the untreated areas. However, when ground sections were attempted, the enamel in all areas was destroyed to such an extent that the method was deemed unsatisfactory.

From teeth protected in a similar manner, ground sections were made immediately after their treatment with silver nitrate. These sections were then treated with 5% lactic acid and the progress of decalcification as evidenced by the formation of bubbles of $CO_2$ was observed through the aid of the microscope. In the process of decalcification, dissolution of the enamel was more rapid in those areas not treated with silver nitrate. The areas treated with silver nitrate floated away *en masse* from the remaining tooth structure as though they were set free by a dissolution of the supporting enamel.

A test was then made to compare the solubility of treated and untreated enamel by the weight-loss of each substance when subjected to acid. For this purpose freshly extracted teeth, relatively free from caries, were used. The crowns were removed from the roots and the coronal portions were mechanically broken up in a steel swedging device. The material was pulverized to pass through a 60 mesh screen, giving a powder of enamel and dentin of nearly uniform size of particles. The enamel was then separated from the dentin by use of bromoform as described by Armstrong and Brekhus (4). This is essentially a separation accomplished by means of their different specific gravities. The enamel was washed with distilled water and dried in an oven at 100°C. to bring to constant weight. This enamel was weighed and divided into two parts. One part constituted the untreated enamel. The other part was treated with a 10% solution of silver nitrate for one hour, after which it was filtered and washed to remove all trace of soluble salts. This sample was then dehydrated and weighed

to determine the weight-gain of silver. This constituted the enamel used as treated material.

Portions of this material were taken so that each sample contained five grams of enamel plus its proportionate weight-gain of silver which was 0.6162 gram. An equal number of five gram samples of untreated enamel were made. Each of these specimens was placed in 100 cc. of 5% lactic acid and allowed to stand. After varying numbers of days ranging from two for the first to twenty-eight for the last, specimens of treated and

TABLE 1

A. The record of the dissolution of untreated enamel when subjected to 5% lactic acid for varying numbers of days.

B. The record of enamel treated with silver nitrate showing the amount of enamel and silver loss when subjected to 5% lactic acid for varying numbers of days.

Original weight of each sample = 5.00008.
Aliquot weight of silver in treated enamel sample 0.6162

| A. UNTREATED ENAMEL | | | | B. TREATED ENAMEL | | | | |
|---|---|---|---|---|---|---|---|---|
| NO. | DAYS ACID | WT. AFTER ACID | ENAMEL LOSS | NO. | WT. AFTER ACID | GROSS LOSS | SILVER LOSS | ENAMEL LOSS |
| | | gm. | gm. | | gm. | gm. | gm. | gm. |
| 1A | 2 | 3.3386 | 1.6614 | 1 | 4.2878 | 1.3284 | 0.2984 | 1.0300 |
| 2A | 4 | 3.0820 | 1.9180 | 2 | 4.4478 | 1.2684 | 0.1953 | 1.0731 |
| 3A | 6 | 3.0070 | 1.9930 | 3 | 4.1142 | 1.5020 | 0.2423 | 1.2597 |
| 4A | 8 | 3.0018 | 1.9982 | 4 | 4.0114 | 1.6048 | 0.1888 | 1.4160 |
| 5A | 10 | 2.9792 | 2.0208 | 5 | 3.9134 | 1.7028 | 0.2060 | 1.4968 |
| 6A | 12 | 2.6285 | 2.3715 | 6 | 3.6886 | 1.9276 | 0.2080 | 1.7196 |
| 7A | 16 | 2.5010 | 2.4990 | 7 | 3.6474 | 1.9688 | 0.1736 | 1.7952 |
| 8A | 20 | 2.4756 | 2.5244 | 8 | 3.5390 | 2.0772 | 0.1564 | 1.9208 |
| 9A | 24 | 2.4924 | 2.5076 | 9 | 3.4759 | 2.1403 | 0.1629 | 1.9774 |
| | 28 | | | 10 | 3.3966 | 2.2196 | 0.1736 | 2.0460 |

untreated enamel were filtered, washed and dried to constant weight. The loss of weight was recorded for each sample. The amount of silver taken into solution by the acid was determined by titrating the filtrate and washings, using Volhard's ammonium-thiocyanate titration method. Correction of the gross loss was then made for the silver found in the filtrate.

Table 1, A is the record of the dissolution of untreated enamel. Table 1, B is the record of treated enamel showing in addition to the net loss of enamel the amount of silver lost by dissolution in the acid. A comparison of the enamel losses in these two series is illustrated in Chart I. The curves

show a marked difference in the rate of dissolution. In the first two days there is a rapid enamel loss of the untreated material, coming to a plateau area in about twelve to sixteen days, after which time dissolution does not materially increase. The curve of the treated material shows a gradual rise in the amount of enamel going into solution and in about twelve to sixteen days it reaches a plateau area which is lower than but almost parallel to that of the untreated material. At no time, however, does the amount of loss of treated material equal that of the untreated material. When ten

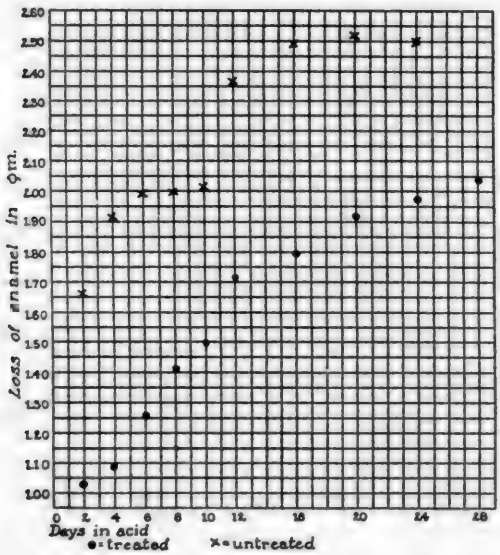


CHART I. A graphic illustration of the behavior of untreated enamel and enamel treated with silver nitrate when subjected to weak organic acid, as recorded in Table 1.

gram samples of untreated enamel were subjected to 100 cc. of lactic acid, the results did not materially differ from the five gram samples. A similar experiment with treated enamel showed a marked increase in early loss of weight, but the final results were comparable to those obtained in the smaller samples.

*Discussion.* Some consideration should be given to the possible combinations of silver nitrate with enamel. Rosebury (5) has shown enamel to contain 0.42% of insoluble organic matter in a form with the

chemical characteristics of keratin and no soluble protein. Assuming that the material used contained its normal organic content, it would be impossible for all the silver reacting to be in combination with organic material. The organic content of a five gram sample would be 0.021 grams. If the combining power of enamel protein were equal to that of serum albumin, which is improbable, the protein in five grams of enamel could combine with approximately 0.0062 gram of silver. If silver proteinate is thus formed, it might decrease solubility of the enamel by the mechanical hindrance offered by this material as suggested by Rickert (3). This might be of greater value in the clinical use of silver than when finely powdered enamel is considered. As it is probable that not more than one-one hundredth of the 0.6162 gram of silver could combine with the organic content, the greater portion must be in combination with the inorganic salts or as free silver. Fluorides and chlorides in the presence of silver, would form chemical combinations which are relatively insoluble and would decrease the rate of dissolution of enamel. These salts, however, are present in such small amounts that their influence would be negligible. The carbonates and phosphates, when combined with silver would form salts which are much less soluble than the carbonate and phosphate salts of sodium and magnesium.

*Relative solubility in cold $H_2O$*

| | | | |
|---|---|---|---|
| $Mg_3(PO_4)_2$.... | .02 | $Ag_3PO_4$..... | .00065 |
| $MgCO_3$ ..... | .0106 | $Ag_2CO_3$..... | .00325 |

| | |
|---|---|
| $Na_3PO_4$ ..... | 28.3 |
| $Na_2CO_3$..... | 7.1 |

Reference to the curve of solubility shown in Chart I shows the untreated enamel to be a readily soluble substance in 5% lactic acid, while the treated enamel acts as a less soluble material. If the decrease in solubility of treated enamel were due to the protection offered by the deposition of free silver only, there should be a slow rise in the first part of the curve with an abrupt upward turn when the acid has penetrated the protected area.

Kanthak (6) reported the solubility of calcium and phosphorus content of enamel. His curves showing the rate of solution of calcium

and phosphorus were structurally similar to the curve for untreated enamel shown in Chart I. The outstanding difference is that when the loss of all the constituents of enamel is considered, the early loss of weight is greater than the loss of calcium and phosphorus. This indicates that the presence of salts more readily soluble than calcium phosphate, such as the magnesium and sodium salts, plays an important part in early decalcification of enamel. The difference in the curves for treated and untreated enamel shows the probability of the conversion of these more readily soluble salts into silver salts which are much less soluble.

## CONCLUSIONS

1. Enamel, after being treated with silver nitrate, becomes more resistant to the action of weak organic acids.

2. Only a small fraction of the amount of silver which combines with enamel can be accounted for by the organic material in the enamel.

3. It is suggested that the reduction in the solubility of enamel in 5% lactic acid after treatment with silver is due principally, if not entirely, to combination of the silver with soluble inorganic material in the enamel so as to form a compound poorly soluble in weak organic acids.

The authors wish to acknowledge the aid of Dr. Ramon F. Hanzal in the chemical aspect of this study.

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# STUDIES IN THE CONSERVATION OF DECIDUOUS AND EARLY PERMANENT TEETH[1]

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This preliminary report of studies now in progress in this department, is based on 352 teeth with deep cavities and exposed pulps treated and recalled for checking at intervals from January, 1930 to January, 1935.

The 352 teeth were divided into four groups according to the treatment indicated, namely: (1) cavity sterilization; (2) pulp capping; (3) pulpotomy; and (4) root-canal therapy. Radiographs were made in all cases during treatment and cavities filled with amalgam. The patients were recalled at various intervals, the teeth in question checked, radiographed, and condition of the gingival tissues and reaction of the teeth to percussion noted. In the first two groups the electric pulp-tester was used to test for vitality. Teeth showing no clinical or radiographic evidence of pathology and which were in function were recorded as successful, while those that showed clinical and radiographic evidence of pathology were recorded as unsuccessful.

The first group (*cavity sterilization*) included 53 deciduous and 78 permanent teeth with deep cavities, in which complete excavation might have resulted in pulp exposures. The cavities were partially excavated, the deepest decay left to avoid exposure, and sterilization attempted with various agents. The agent used in most cases was ammoniacal silver nitrate followed by eugenol. About 87 per cent of the deciduous cases returned for checking; of these 89 per cent were successful and 11 per cent unsuccessful. Of the permanent teeth 82 per cent returned for checking; of these, 97 per cent were successful and 3 per cent unsuccessful. More than half of the total successful

[1] A paper on the methods of treatment used in these studies was read by the author before the Ninth District Dental Society, New York, on April 13, 1935. Some parts of this work were included in a paper by L. R. Stowe, and S. N. Rosenstein read before the International Association for Dental Research, New York Section, May 23, 1935.

cases were retained in position and function for from one and one half to four years.

The second group (*pulp capping*) included 144 deciduous and 30 permanent teeth, with pulps actually exposed by caries or trauma, and both sensitive and hemorrhagic at the point of exposure. These cases were completely excavated, phenolized and treated with a capping material, such as zinc oxide-eugenol, a thymol-zinc oxide preparation, or cement powder mixed with cement liquid previously saturated with silver nitrate,[2] and then filled. Of 61 per cent of the deciduous cases which returned for checking, 80 per cent were successful and 20 per cent unsuccessful. Of the permanent cases 63 per cent returned for checking, of which 95 per cent were successful and 5 per cent unsuccessful. In about half of the returned successful cases, the teeth were retained in service from one to about four years.

The third group (*pulpotomy*) consisted of 20 deciduous teeth in which there was fairly extensive destruction by caries with degeneration of part of the coronal portion of the pulp. In each case the chamber was opened and the coronal portion of the pulp removed, the area cauterized and a layer of pulp-chamber filling inserted and covered with cement. Fillings were subsequently placed in all teeth. Of the various agents used in this group, Gurley Paste appears to be the most suitable. Of 15 cases that returned for checking, 11 were successful and 4 unsuccessful. The successfully treated teeth were retained for varying periods up to 33 months.

The fourth group (*root canal therapy*) comprised 27 deciduous teeth, in which the pulps were almost entirely degenerated. Putrescent pulps with fistulae or with radiographic evidence of pathological involvement at root ends or root bifurcations were not included. In the treated teeth, the chambers and orifices of the canals were carefully cleansed, dressings of creosote or formocresol used for several treatments and the canals filled with root-canal paste and covered with cement. The cavities were filled at subsequent visits. The most frequently used root-canal paste was Gysi Paste. Of this group 22 reported for checking; 13 of these were successful and 9 unsuccessful.

[2] A report on the use of this material in pulp capping in deciduous teeth was read by L. R. Stowe before the Faculty and Staff of the School of Dental and Oral Surgery of Columbia University, February 13, 1935.

The successful teeth were kept in service for periods up to about two and a half years.

The high percentage of success in the cavity sterilization and pulp-capping groups indicates that the deciduous pulp can well withstand the irritation of deep decay and exposure, can be kept vital, and by these methods of treatment the period of tooth-service can be prolonged several years until exfoliation. While the pulpotomy and root-canal-therapy groups do not show as high a degree of success as the first two groups, the studies indicate that a majority of the treated teeth requiring radical procedures can be maintained in normal position and function for appreciable lengths of time.

All the teeth included in these studies were treated by students under the supervision of the staff of the Division of Dentistry for Children, with the exception of several cases treated by Dr. B. Nathanson, Clinical Assistant.

A further report will be made at a future date, covering more cases and giving more detailed information.

# A STATISTICAL ANALYSIS OF DENTAL PERIODICALS[1]

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Most methods for judging professional periodicals are subjective, involving the preferences or interests of the judge and a comprehensive knowledge of the subject matter. An objective method was used to determine with some probability which of the present dental periodicals are of sufficient value to be placed on the reference shelves of a dental library.

One way of estimating the *quality* of a paper may be by counting the number of references cited by the author. The *usefulness* of any given periodical to the profession may be estimated by the number of times it is quoted from or referred to in the literature of the profession.

A group of students[2] counted the references in certain volumes (table 1) of 21 dental periodicals. A total of 21,901 references were counted mentioning 1633 different periodicals. In order to obtain comparable figures, the files of approximately the same period were taken (1915–1933). Two volumes not available are so indicated.

In table 1 is shown the number of references (r) to periodical literature. These numbers divided by the number of volumes (v) give the fractions (r/v) which show the wide variation of 302.8 references per volume for *The Journal of the American Dental Association*, to 5.8 references per volume for *Dental Rays*. These ratios may be conveniently grouped into three divisions having approximate averages of 250, 115 and 30 references per volume, respectively. To indicate the scope of the literature quoted, the last column gives the number of different periodicals mentioned. *The Journal of the American Dental Association* mentions in its 20 volumes nearly 500 different periodicals, while *The Dental Student's Magazine* only 17.

[1] Read at a meeting of the San Francisco Section, International Association for Dental Research, February 28, 1935. See also proceedings of the Association: *J. Den. Res.*, *15*, 215 (1935).

[2] This work was conducted as an S.E.R.A. project.

A list of the periodicals most frequently referred to is given in table 2. There are several factors which influence the frequency with which a journal is mentioned; one being distribution. A periodical having a circulation of 35,000 copies is obviously better known and

TABLE 1

*Referring periodicals: Showing number of references in periodicals examined*

| NAME | VOLUMES EXAMINED | NUMBER OF REFERENCES | REFERENCES | | PERIODICALS MENTIONED |
|---|---|---|---|---|---|
| | | | Per volume | Average per group | |
| | v | r | r/v | r/v | |
| 1. *J. A. D. A.* | 1–20 | 6057 | 302.8 | | 496 |
| 2. *Curr. Res. Anest. Analg.* | 3–12 | 2420 | 242.0 | 251.4 | 455 |
| 3. *J. Periodontology* | 2–3 | 419 | 209.5 | | 133 |
| 4. *Int. J. Orthodont.* | 2–19[1] | 2519 | 125.9 | | 452 |
| 5. *D. Cosmos* | 57–75 | 2326 | 122.4 | | 259 |
| 6. *D. Outlook* | 3–12 | 2420 | 119.2 | 115.6 | 455 |
| 7. *J. D. Research* | 1–13 | 1425 | 109.6 | | 351 |
| 8. *Illin. D. J.* | 1–2 | 202 | 101.0 | | 54 |
| 9. *D. Survey* | 6–9 | 358 | 89.5 | | 112 |
| 10. *D. Items Int.* | 37–55 | 955 | 50.3 | | 173 |
| 11. *Am. D. Surg.* | 46–50 | 223 | 44.6 | | 109 |
| 12. *D. Summary* | 37–44 | 356 | 44.5 | | 97 |
| 13. *D. Digest* | 21–39 | 584 | 30.7 | | 106 |
| 14. *Oral Hygiene* | 1–22[2] | 627 | 29.9 | | 121 |
| 15. *Oral Health* | 9–22 | 331 | 23.6 | 29.4 | 158 |
| 16. *Pac. D. Gazette* | 24–41 | 303 | 16.8 | | 128 |
| 17. *Apollonian* | 1–7 | 109 | 15.5 | | 54 |
| 18. *Calif. S. D. J.* | 1–9 | 110 | 12.2 | | 52 |
| 19. *D. Students Mag.* | 5–10 | 68 | 11.3 | | 17 |
| 20. *Ohio S. D. Assn.* | 1–6 | 48 | 8.0 | | 26 |
| 21. *D. Rays* | 1–7 | 41 | 5.8 | | 21 |
| Total | | 21,901 | — | | 1633 |

[1] Exclusive of volume 8. [2] Exclusive of volume 4.

more frequently quoted than one having an issue of only a few hundred. *Oral Hygiene* with an issue of 65,000 copies has apparently the largest circulation, hence it is high in proportion to its relative value.

The *percentage* indicates the frequency with which the periodical is referred to, for example, *The Dental Cosmos* is given 4173 times in the

total of 21,901 references (thus, approximately 20% of the references are to *The Dental Cosmos*). The column q/v gives the number of references per volume. These ratios likewise can be conveniently

TABLE 2

*Quoted periodicals: Showing number of times dental periodicals referred to*

| NAME | NO. OF VOL. TO 1933 | NUMBER TIMES REFERRED TO | PER CENT OF TOTAL | REFERRED TO | |
|---|---|---|---|---|---|
| | | | | Per volume | Average per group |
| | v | q | % | q/v | q/v |
| 1. *J. A. D. A.* | 20 | 2937 | 13.41 | 146.85 | |
| 2. *D. Cosmos* | 75 | 4173 | 19.0 | 55.64 | 82.1 |
| 3. *J. D. Research* | 13 | 569 | 2.59 | 43.8 | |
| 4. *Int. J. Orthodont.* | 19 | 494 | 2.25 | 26.0 | |
| 5. *Brit. D. J.* | 15 | 305 | 1.40 | 20.3 | 18.5 |
| 6. *Oral Hygiene* | 24 | 377 | 1.72 | 15.7 | |
| 7. *D. Items Int.* | 55 | 660 | 3.01 | 12.0 | |
| 8. *D. Summary* | 44 | 241 | 1.10 | 5.4 | |
| 9. *D. Digest* | 40 | 213 | 0.97 | 5.3 | |
| 10. *Pac. D. Gazette* | 41 | 175 | 0.80 | 4.3 | 4.4 |
| 11. *D. Outlook* | 20 | 82 | 0.37 | 4.1 | |
| 12. *D. Record* | 52 | 152 | 0.65 | 2.9 | |

*Showing number of times foreign, medical or scientific periodical referred to*

| Foreign periodicals | q | Medical and scientific periodicals | q |
|---|---|---|---|
| *Zeit. f. Stomatol.* | 203 | *J. A. M. A.* | 1461 |
| *Revue Stomat.* | 99 | *Lancet* | 332 |
| *D. Monatschr.* | 89 | *J. Biol. Chem.* | 205 |
| *Zahnaerztl. Rundsch.* | 88 | *J. Infect. Dis.* | 201 |
| *D. mediz. Wochenschr.* | 72 | *N. Y. Med. J.* | 190 |
| *Viertelj. Zahnheilk.* | 71 | *Brit. Med. J.* | 184 |
| *Compt. rend. Soc. Biol.* | 56 | *Surg., Gyn. & Obstr.* | 168 |
| *Fortschr. Zahnh.* | 54 | *Arch. Int. Med.* | 154 |
| | | *J. Biochem.* | 137 |
| | | *J. Exp. Med.* | 136 |
| | | *Am. J. Dis. Childr.* | 128 |

divided into three groups, having approximate averages of 80, 20 and 4 references per volume, respectively.

The fact that *The Journal of the American Dental Association* is referred to nearly 3.5 times as frequently as the *Journal of Dental*

*Research* is probably due to the more limited distribution of the latter. Of foreign periodicals, the German are much more frequently referred to than the French. Of medical periodicals, *The Journal of the American Medical Association* leads; while of scientific periodicals, the chemical literature is most frequently quoted. The wide interest of dental science is indicated by the fact that the list of 1633 periodicals contains titles of a ceramic, industrial, homeopathic, historical, anthropological, veterinary, botanical, biophysical, zoological and pharmaceutical nature.

# EVIDENCE CONCERNING A LARGE FOSSIL HYRAX

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In 1892 Lydekker (1) stated "it is somewhat remarkable that at present no extinct animals have been discovered which appear allied to the hyraces." Some years later remains of such animals were discovered but even as late as 1925 Zittel (2) records that fossil forms of the family *Hyracidae* (*Procaviidae*) are not known. Since in the literature available there is no record of discoveries since 1925, this report concerning fossilized remains of the family discovered by the writer seems warranted.

The remains were found on the farm Sterkfontein, some 26 miles from Johannesburg and about 1 mile distant from the Sterkfontein Lime Works where in the autumn of 1936 Dr. Robert Broom recovered a new fossil ape which he named *Australopithecus transvaalensis*. They consist of the right half of a mandible with all the incisor, premolar and molar teeth fully erupted and excellently preserved and part of a skull with three molar and three premolar teeth in situ on the left side of the palate. As will be seen (Fig. 1), the outer or buccal surface of the mandible is imbedded in rock. The inner or lingual surface together with the teeth were similarly imbedded but the limestone covering was removed exposing and clearly defining the lingual surface of the bone and all the surfaces of the teeth. The skull and maxillary teeth are embedded in a second rock but, with the exception of the third molar tooth, are so ill preserved and fragmentary that they do not lend themselves to detailed description and measurement.

Too little is known concerning tertiary and recent rock formations in South Africa to permit any definite statement concerning the geological age of the rocks in which the remains are preserved but it seems that it is ancient, probably from the middle pleistocene and possibly from a much earlier period.

According to Thomas (3) there are fourteen species of hyraces all of which belong to a single genus, *Procavia*. Only two species are found in South Africa, (a) *P. arborea* and (b) *P. capensis*. The former has small rounded teeth of the brachyodont type while in *P. capensis* the teeth are larger and of hypsodont type.

The teeth in the fossil remains are of hypsodont type and in their general form closely resemble those of *P. capensis*. They are however, considerably larger; in addition the mandibular series exhibits features not present in the specimens of *P. capensis* in our Museum. The skull and mandible are also larger.

FIG. 1—Note difference in size of right half of fossil mandible and corresponding half of mandible of *P. capensis*.

There are six specimens of *P. capensis* in our Museums of Odontology and Anatomy. These have been measured and their stage of development estimated according to the methods recommended by Thomas. The result is shown in Table 1.

It is considered improbable that the mandible belongs to the skull, since, when found, the two rocks containing these fossils were separated by a considerable distance. There is no doubt that both fossils belong to the same species of *Procavia* and, as is shown by the measurements in Table 1, this species was markedly larger than the living South African hyrax, *P. capensis*. It was not only bigger than *P. capensis* but, according to the measurements provided by Thomas, larger than any existing species of *Procavia*. Lydekker states that

TABLE 1

| | CATALOGUE NUMBER | STAGE OF DEVELOPMENT | LENGTH OF | | HEIGHT OF MANDIBLE AT M1 | LABIO-LINGUAL WIDTH OF | | HEIGHT OF CROWN OF M3 |
|---|---|---|---|---|---|---|---|---|
| | | | Skull | Mandible[1] | | Max. M1 | Man. M1 | |
| | | | *mm.* | *mm.* | *mm.* | *mm.* | *mm.* | *mm.* |
| Skulls of *P. capensis* | A1. | VIII | 83.0 | 75.0 | 16.0 | 7.5 | 4.0 | |
| | A2. | VIII | 82.0 | 75.0 | 15.0 | 7.5 | 4.5 | |
| | 1143. | VIII | 83.0 | 75.0 | 15.5 | 8.0 | 4.5 | |
| | 908. | VII | 79.0 | 70.0 | 15.0 | 7.0 | 4.0 | |
| | D.A1. | V | 72.0 | 68.0 | 15.0 | 7.0 | 4.0 | |
| | A3. | IV | 72.0 | 68.0 | 15.0 | 7.0 | 4.0 | |
| Fossil | F.D.A. 6 Skull | VIII | 115.0 *approx.* | — | — | 10.5 *approx.* | — | 7.0 (worn) |
| | F.D.A. 8 Mandible | VIII | — | 104.0 | 25.0 | — | 6.0 | 7.0 |

[1] The length of the mandible was measured from between the first incisor of the right side to the distal border of the ramus of the mandible of the same side. This measurement was taken to permit comparison with the fossil mandible.

*P. Shoana* is the largest of the hyraces and except for two specimens of *P. dorsalis* which have basal lengths of over 100 mm., Thomas confirms this statement. In one species of *P. dorsalis* reported upon by the latter the basal length was 100 mm. while in another it was 113 mm. As Thomas stated, "this is one of the few species that is quite distinct from all its neighbours . . . its proportionately large head and small body distinguish it at once from its allies." In addition, it is distinguished from *P. capensis* by its short crowned teeth.

The first mandibular premolar is present in the fossil mandible. According to Thomas it is very unusual to find this tooth present in

adults with hypsodont teeth, e.g. *P. capensis*, *P. abyssinica*. It is absent in all the specimens in our Museum with the exception of D.A1. It is of very small size, measuring only 3.0 mm. mesiodistally, and in having only one root supports Thomas' statement that in species with hypsodont teeth two roots are not developed. In this connection, it must be pointed out that in our specimen D.A1. two very distinct roots are present on this tooth on one side of the lower jaw—the tooth on the opposite side is missing. Another and very conspicuous difference between the fossil mandible and the mandibles of *P. capensis* in our Museum, is the relative size of the first and second incisors. In all our specimens of *P. capensis* the first mandibular incisor is distinctly smaller than the second incisor. In the fossil these two teeth are of equal size and thus it would appear that in the modern hyrax the first is tending to be eliminated from the dentition.

Although the form of both the skull and teeth of this fossil hyrax are similar to the corresponding parts in the living *P. capensis* yet their large size coupled with a difference in the relative proportion of the lower incisor teeth and the presence of the first mandibular premolar suggests that the animal requires specific designation. Accordingly I propose the name *Procavia transvaalensis, sp. nov.*

I have to thank Mr. Barlow of the Sterkfontein Lime Works for his kindness in permitting me to remove this and other fossilized material from the farm. I have also to express my gratitude to Messrs. Julius Staz, M. Monk and P. Orange for assisting me to collect material and for other valuable services in connection with removing the remains from the rock and preparing this report.

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# A STUDY OF THE BACTERICIDAL, BACTERIOSTATIC AND PEPTIZING ACTION OF CERTAIN DENTIFRICES[1]

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In the entirely healthy and normally-functioning mouth the activity of the tongue, lips, cheeks and the whole of the masticatory apparatus as well as the solvent action of the saliva and certain ferments and non-pathogenic bacteria, play important rôles in maintaining mouth health.

Where these natural aids have been impaired and are no longer sufficient, artificial ones must be called into play. More and more such mechanical and chemical assistances as dentifrices may provide, are being utilized. That dentifrices do not appreciably decrease the bacterial content of the mouth has been pointed out by Leonard and Feirer (1, 2); a finding confirmed by our experiments with a variety of the most popular commercial tooth pastes and powders. On the other hand there is strong indication that in some instances a definite bacteriostatic or inhibitory effect does exist.

Such organisms as the streptococci, staphylococci, bacillus fusiformis and the spirillum of Vincent and Plaut may be constantly found in the mouth both in disease and in health. The use of a germicide only temporarily inhibits these organisms; they will return again in great numbers and in a comparatively short time.

Thus it might be argued that there is no advantage in using any antiseptic mouth-wash since it accomplishes no permanent results. Against this argument, the investigations of Thompson and other members of the Georgetown Research Group (3), with respect to the organisms in ulceromembranous stomatitis (Vincent's), showed that the mere presence of the spirillum (*Borrelia Vincenti*) and the *bacillus fusiformis* did not constitute a diseased condition. Only when the

[1] This study was made possible by a grant from the William S. Merrell Company.

organisms were present in extremely large numbers could the clinical signs of this disease be recognized and in every case where the clinical signs were positive, the organisms were present in large numbers. The control of this disease seemed to resolve itself into a plan of treatment intended to keep down the *number* of organisms.

While the present study indicates that certain types of dentifrices may be of value in accomplishing this task, the frequently exaggerated and misleading claims of manufacturers has made it necessary for Dentistry and its allied professions to establish a rational and honest basis for the selection of these artificial aids to mouth health. A considerable amount of work has been done in this field, yet the subject is by no means a closed one and still further studies are indicated.

Carroll of the Georgetown research group (4) made a systematic study of the bactericidal qualities of a number of the better known and more widely sold dentifrices. Phenol coefficients (after the method of the Food and Drug Administration (5)) were determined for the different products under consideration, and mouth rinsings with comparable dilutions of the drugs were plated out under identical conditions and bacterial (colony) counts were made of these plates.

The more important of Carroll's conclusions were as follows:

"In the laboratory tests a surprisingly large number of widely-advertised mouth-washes were found to have a very low phenol coefficient: only a few of the pastes and powders showing any bacteriostatic properties."

"With the single exception of hexylresorcinol (which also was possessed of the highest phenol coefficient[2]) all the liquids having bactericidal properties which were tested, proved irritating to the tissues of the mouth when used in the dilution necessary to produce bactericidal effects."

"While hexylresorcinol is bactericidal in the test-tube, and sodium ricinoleate is not generally so, still in these experiments less organisms were found in the mouth after the use of sodium ricinoleate, and the counts remained lower for a longer period than when hexylresorcinol was used."

These apparently paradoxical results seemed to demand further analysis, and a thorough comparison of these two reagents was instituted. The present study may be divided into three experiments.

In the first experiment ten individuals were used, each subjected to exactly the same procedure which follows:

[2] The statement in parentheses was inserted by the present author, S. V. M.

1. After lunch the person expectorated into a sterile tube. This saliva in various dilutions was plated on plain agar medium as rapidly as possible.

2. The individual was instructed to brush his teeth (Charter's method) for two minutes using a paste dentrifrice containing sodium ricinoleate. After brushing, a rinse of one minute was required, using a sodium ricinoleate mouth wash. Immediately after the process was completed, expectorated saliva was placed in a sterile test tube and plated as in the first step.

3. One, two and three hours later saliva was collected and plated in the first step.

4. All plates were incubated for 48 hours at 37.5° and colonies counted with a hand lens.

Each individual went through the procedure three times at two-day intervals and the average of the three counts was taken for each of the five tests. The entire process was then repeated using distilled water as a control instead of a mouth wash.

In each case the average count from step 1 (that taken directly after lunch) was used as 100%. From this basis the percentage of increase or decrease in bacterial counts in the remaining steps was computed. These results are shown in Figs. 1 and 2.

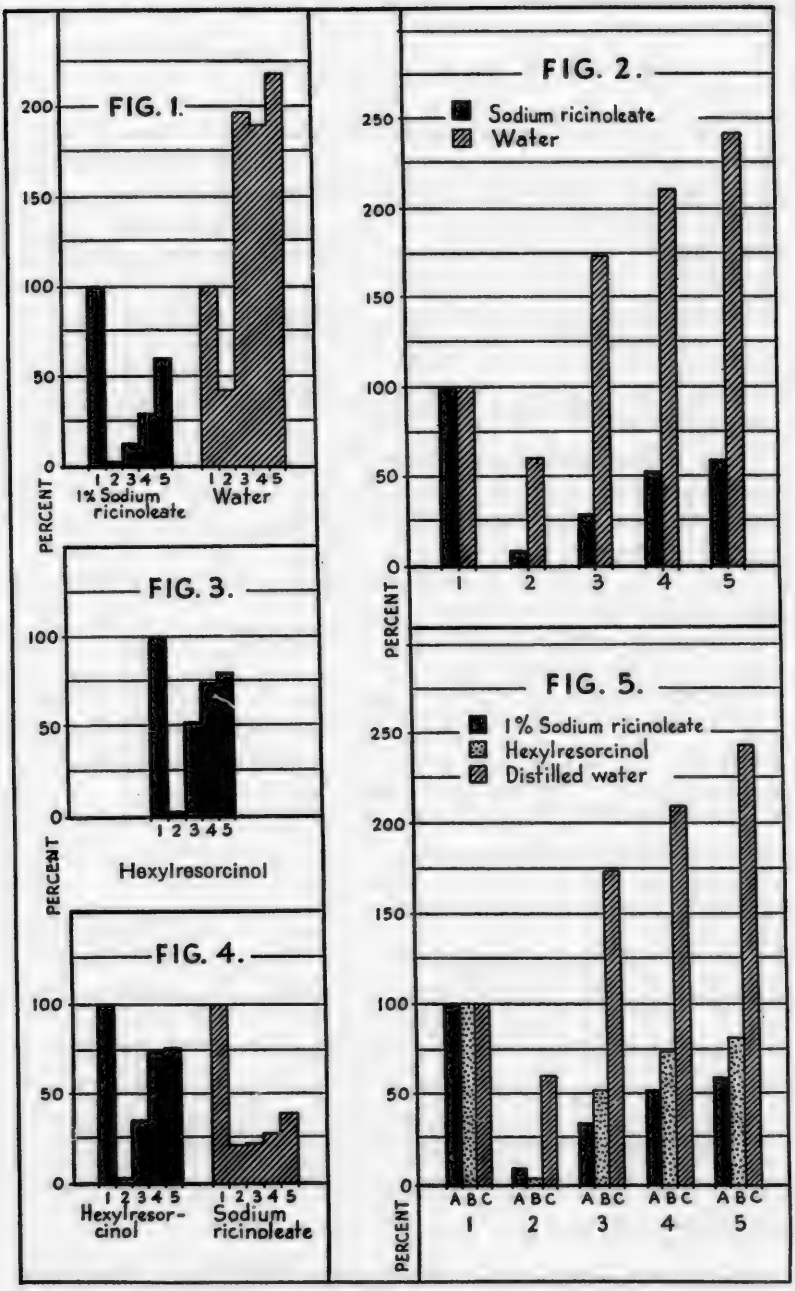
It will be seen readily that the individuals who have used the sodium ricinoleate mouth wash have a materially decreased bacterial count for a considerable period after such usage, as compared with the studies where distilled water only has been used for a rinse.

The following table of percentage increase or decrease in bacterial count based on the averages of the total number of cases studied shows how marked is this contrast.

| Water Rinse | | Sodium ricinoleate 1.0% Rinse |
|---|---|---|
| 100% | After Lunch | 100% |
| 61.9% | After 1st Rinse | 8.1% |
| 174.7% | 1 hr. after 1st Rinse | 24.4% |
| 210.6% | 2 hrs. after 1st Rinse | 53.2% |
| 242.4% | 3 hrs. after 1st Rinse | 58.8% |

This experiment revealed very clearly the effect of sodium ricinoleate as contrasted with distilled water, and carried out the suggestion of the previous studies that the product was possessed of definite bacteriostatic qualities.

In the second experiment a similar group of individuals was subjected to the identical routine as in the previous experiment, except that a hexyl-




FIGS. 1–5. Column 1 represents the first total count, after lunch and before using mouth wash (arbitrarily taken as the standard of comparison, or 100%).

Subsequent counts were made and calculated on this scale, *i.e.*, in Fig. 1, suppose that column 1 represents a count of 100,000 organisms per cc. of saliva and is taken as 100%. The next, column 2, taken immediately after using the sodium ricinoleate rinse shows a count of approximately 100 or 1%; column 3—approximately 10,000 or 10%, etc.

resorcinol paste and a hexylresorcinol mouth wash were used instead of those containing sodium ricinoleate. A typical graph of this series is shown in Fig. 3. In Fig. 4 is shown graphically a comparison between the effects of sodium ricinoleate and hexylresorcinol when used on the same individual at two day intervals between the tests. The averages of all the cases of tests are shown graphically in Fig. 5. It is quite apparent that the bacteriostatic action of sodium ricinoleate is far greater, although hexylresorcinol is possessed of a higher phenol coefficient.

In attempting an explanation of the phenomenon, several theories were advanced and tested. For example it seemed that sodium ricinoleate may be possessed of some unique chemicophysical cleansing qualities and peptizing powers which cause so complete a removal of the free organic matter in the mouth that virtually nothing is left to act as a culture medium for microörganisms.

In the third experiment another similar group of individuals was used to compare the number and size of the colloidal particles removed from the mouth (an index of the cleansing power) by rinsing with the respective products being studied. Each subject rinsed his mouth with distilled water (as a control) and the rinsings were photographed by dark-field illumination. Then the process was repeated using rinses containing, first, sodium ricinoleate and second, hexylresorcinol. After a two-day interval this process was repeated, except that the hexylresorcinol rinse followed the distilled water rinse, and in turn was followed by the sodium ricinoleate rinse, using the same strength solutions. Typical photographs from this series are shown in Figs. 6 to 10 inclusive. It was found that invariably, by far the larger number of colloidal particles were present in the sodium ricinoleate rinsings, whether its use preceded or followed the hexylresorcinol. The hexylresorcinol and distilled water were about comparable in their effects.

In order to test the respective reagents for peptizing qualities, six clean test tubes were taken and into each 1 cc. of 5% egg albumen was introduced. In addition, to five tubes, 1 cc. of decreasing strengths of a sodium ricinoleate solution was added as follows: 1.0, 0.75, 0.50, 0.25, 0.125 per cent. To the sixth was added 1 cc. of distilled water. In another similar series identical strengths and quantities of hexylresorcinol solution were added to the albumen. All of the tubes were placed in a waterbath at 65°C. for five minutes. The results are shown in the photographs in Figs. 11 and 12. It is apparent that even the weakest solution of the sodium ricinoleate was

sufficient to completely peptize the albumen, while the strongest solution of the hexylresorcinol was not.

These studies on the bacteriostatic qualities of sodium ricinoleate point to an analogous condition known to surgeons with regard to asepsis of the hands. They have had well demonstrated that the most effective method is thorough *cleansing*, rather than reliance on the use of chemical bactericides. The same situation seems to govern mouth prophylaxis.

## SUMMARY

Sodium ricinoleate, although not possessed of bactericidal qualities, as evidenced by its phenol coefficient, when used as a mouth wash distinctly lowers the bacterial count and holds it at a low level for a considerable period. These qualities are in marked contrast to hexylresorcinol, although it has a higher phenol coefficient.

Sodium ricinoleate apparently is possessed of unique cleansing powers as well as a peptizing action, which so thoroughly cleanse the oral tissues that little or no organic material is permitted to remain in the mouth to act as a culture medium for mouth organisms.

The author wishes to express his appreciation to Dr. M. Mollari and Robert J. Reedy of the Bacteriology Department, Georgetown University, for their help and cooperation. Special credit is due Drs. Bryant S. Carroll and Edw. L. Thompson for original research at the same institution. Dr. T. H. Rider suggested the peptizing experiment as used. For the photographic work I am indebted to Mr. A. E. Alderman.

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FIG. 6. Case R. a. Dark-field microphotograph (×915) of distilled water after rinsing mouth for one minute.

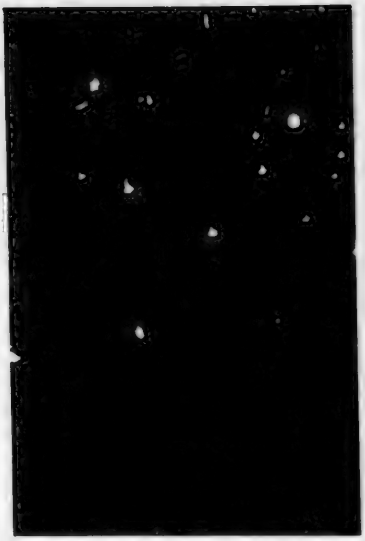

FIG. 6. b. Dark-field microphotograph (×915) of hexylresorcinol solution after rinsing mouth for one minute. Same case, immediately following rinse with distilled water shown in 6a. Note how few additional particles are removed.

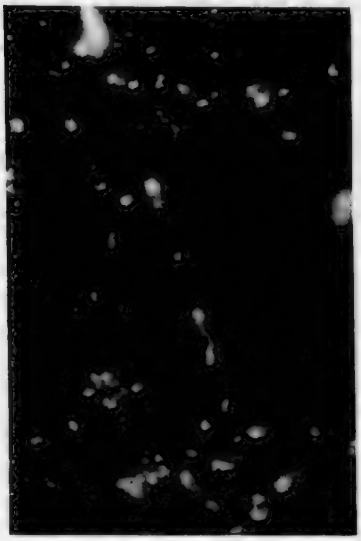

FIG. 7. Case S. a. Dark-field microphotograph (×915) of distilled water after rinsing mouth for one minute.

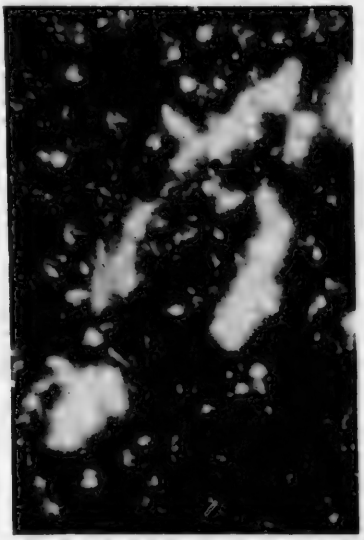

FIG. 7. b. Dark field microphotograph (×915) of sodium ricinoleate after rinsing mouth for one minute. Same case, immediately following rinse with distilled water shown in 7a. Note numerous particles. Contrast with 6b.

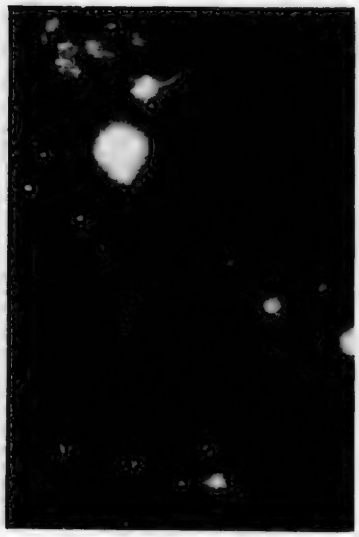

FIG. 8. Case T. a. Dark field microphotograph (×915) of hexylresorcinol after rinsing mouth for one minute. Immediately following rinse with distilled water. Relatively few particles.

FIG. 8. b. Dark-field microphotograph (×915) of sodium ricinoleate after rinsing mouth for one minute. Same case, immediately following rinse with hexylresorcinol shown in 8a. More particles still present than shown in hexylresorcinol rinse.

FIG. 9. Case U. a. Dark-field microphotograph (×915) of sodium ricinoleate after rinsing mouth for one minute. Immediately following rinse with distilled water. Note numerous particles removed by ricinoleate.

FIG. 9. b. Dark-field microphotograph (×915) of hexylresorcinol after rinsing mouth for one minute. Same case, immediately following rinse with sodium ricinoleate shown in 9a. Note how few remaining particles are removed. Contrast with Fig. 9.

FIG. 10. Case V. a. Dark-field microphotograph (×915) of distilled water after rinsing mouth for one minute.

FIG. 10. b. Dark-field microphotograph (×915) for ricinoleate after rinsing mouth for one minute. Same case, immediately following rinse with distilled water shown in 10a. Note numerous large particles removed by ricinoleate wash even though mouth was just washed by water.

FIG. 10. c. Dark-field microphotograph (×915) of distilled water after rinsing mouth for one minute. Same case, immediately following rinse with ricinoleate and previously with water as shown in Fig. 10a. and b. Note diminished number of particles removed.

FIG. 11a. Hexylresorcinol—before heating. Each tube contains 1 cc. of egg albumen. Tube 1. Contains 1 cc. of 100% hexylresorcinol. Note cloudiness. Tube 2. Contains 1 cc. of 75% hexylresorcinol. Tube 3. Contains 1 cc. of 50% hexylresorcinol. Tube 4. Contains 1 cc. of 25% hexylresorcinol. Tube 5. Contains 1 cc. of 12½% hexylresorcinol. Tube 6. Contains 1 cc. of distilled water. Control. Note regularly decreasing turbidity of solutions, paralleling decreasing strengths of the hexylresorcinol.

FIG. 11b. Same tubes after heating at 65° for five minutes in water bath. Note consistent coagulation in all tubes, including control. No peptizing effect evidenced.

FIG. 12a. Sodium ricinoleate—before heating. Each tube contains 1 cc. of egg albumen. Tube 1. Contains 1 cc. of 1.0% sodium ricinoleate. Tube 2. Contains 1 cc. of .75% sodium ricinoleate. Tube 3. Contains 1 cc. of .50% sodium ricinoleate. Tube 4. Contains 1 cc. of .25% sodium ricinoleate. Tube 5. Contains 1 cc. of .12½% sodium ricinoleate. Tube 6. Contains 1 cc. of distilled water. Control.
Note how turbidity in this series increases slightly as strength of reagent decreases.

FIG. 12b. Same tubes after heating at 65°C. for five minutes in water bath. Note marked clearness of all tubes except control. Complete peptization, even in weakest solution of sodium ricinoleate.

# VARIATIONS IN THE LESIONS AND ACTIVITY OF DENTAL CARIES[1]

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## INTRODUCTION

It is a common clinical observation that the activity of dental caries varies throughout the different periods of life. Statistics indicate that dental destruction is most active during youth and up to the age of forty. After this time dental caries becomes less and less active and in many persons ceases entirely. Still later, at about the age of fifty or sixty, the teeth of some persons are again affected.

The purpose of this paper is to classify dental caries according to the types of lesions and the ages at which they are commonly found. This information may aid in a better comprehension of the mechanism of tooth destruction. In addition to this classification, a changing physical characteristic of the teeth will be discussed which may explain, to a great extent, the reason for the age variations in dental caries.

## CLASSIFICATION OF THE LESIONS OF DENTAL CARIES

A classification of the lesions of dental carries now seems worthwhile as our concept of this condition is changing. The old view that the destruction of the teeth was due solely to food retention and bacteria is giving way to a more complex conception of the problem. Evidence (1) for instance, that teeth have a variable resistance to attack, is seen in the decay of only one of two closely adjoining teeth. Further, the fact that an increased vitamin D content in the diet of children reduces the activity of dental caries, indicates that systemic conditions, either by way of the saliva or the dental pulp, or both affect the welfare of the teeth.

The lesions of caries attacking the teeth vary in form, location and rapidity of penetration; they can be classified into: (1) acute crown caries;

[1]Presented at the Thirteenth General Meeting of the International Association for Dental Research, Chicago, March 16–17, 1935; *J. Den. Res.*, 15: 209, 1935.

(2) chronic caries and (3) acute root caries. Erosion, which affects the cervices of the teeth, is not included, as the mechanism of this destructive process seems to be different than that active in dental caries.

The illustrations included in this paper are diagrams which include observations of a large number of sections of teeth. It is believed that these sketches will illustrate more clearly than photomicrographs the cardinal

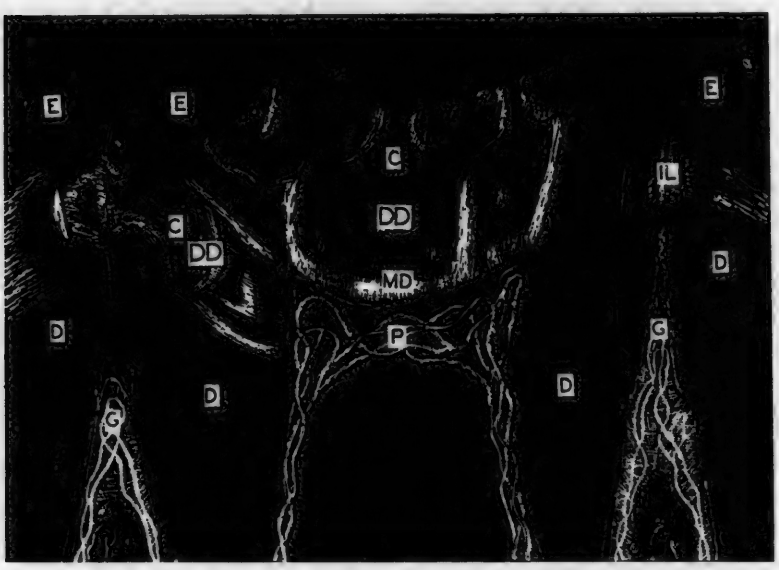


FIG. 1. ACUTE DENTAL CROWN CARIES

This type of lesion destroys rapidly the teeth of young persons due, partially to their high permeability. Three advanced lesions are shown on the occlusal surface and one on the approximal surface of a lower molar. The entrance of each cavity (C) is small, permitting undisturbed food retention; the layer of decalcified dentin (DD) is thick; the dentin (MD) separating the cavity from the dental pulp (P) has been only slightly modified; allowing a rapid penetration of the lesion. No secondary dentin has been formed, so that the entire tooth is highly permeable. The pulp is very vascular. D = dentin; E = enamel; IL = incipient lesion; G = gingival papilla.

differences in the three types of lesions of dental caries. A great number of photomicrographs would be necessary to illustrate this classification.

(1) *Acute dental crown caries* affects principally children and young adults. The entrance of the cavity or lesion (*fig. 1, C*) remains small for a considerable time after its inception and the destructive process is rapid, soon involving a large part of the interior of the crown. The cavity is lined with a layer of decalcified dentin (*DD*), particularly extensive at its

base. Modified dentin (*MD*) is noted separating the cavity from the dental pulp. This zone is not yet carious but its tubular structure has been affected throughout its entire thickness (*fig. 1*). The penetration of the acute lesion of dental carries is usually so rapid that the pulp (*P*) has no

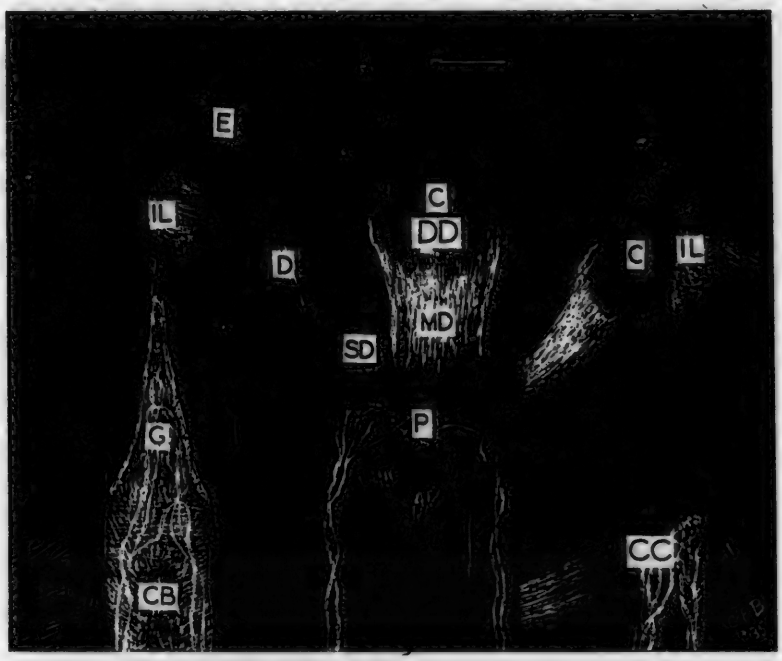


FIG. 2. CHRONIC DENTAL CARIES

This type of lesion progresses slowly and is most common in adults. The entrance to the cavity (C) is large, giving less opportunity for undisturbed food retention; the layer of decalcified dentin (DD) is thin; the permeability of the modified dentin (MD) as well as that of the entire crown is greatly reduced due to changes in its tubules and the formation of secondary dentin (SD) in the pulp chamber (P). The root of the tooth, on the other hand, often retains its original permeability due to the lack of formation of secondary dentin in the pulp canal. Loss of approximal contact allows food impaction, resulting in the recession of the gingival papilla (G); thus the neck of the tooth is exposed to cervical caries (CC). The vascularity of the pulp (P) is less than in the teeth of young persons. D = dentin; E = enamel; IL = incipient lesion; G = gingival papilla; CB = crest of alveolar bone.

opportunity to shield itself by laying down secondary dentin. Acute dental crown caries affects first the enamel fissures or faults of the maxillary and mandibular molars and premolars, later the approximal surfaces of these teeth; then the maxillary incisors and canines, and lastly the anterior mandibular teeth. The appearance of carious lesions on the lower anterior

teeth is usually a sign of rampant dental caries. If the cervical areas of any teeth become affected, particularly when this occurs before middle life, it indicates also a high activity of dental caries.

(2) *Chronic dental caries* affects the various surfaces of the teeth in the same order as does that of the acute type, but its severity is lower. The

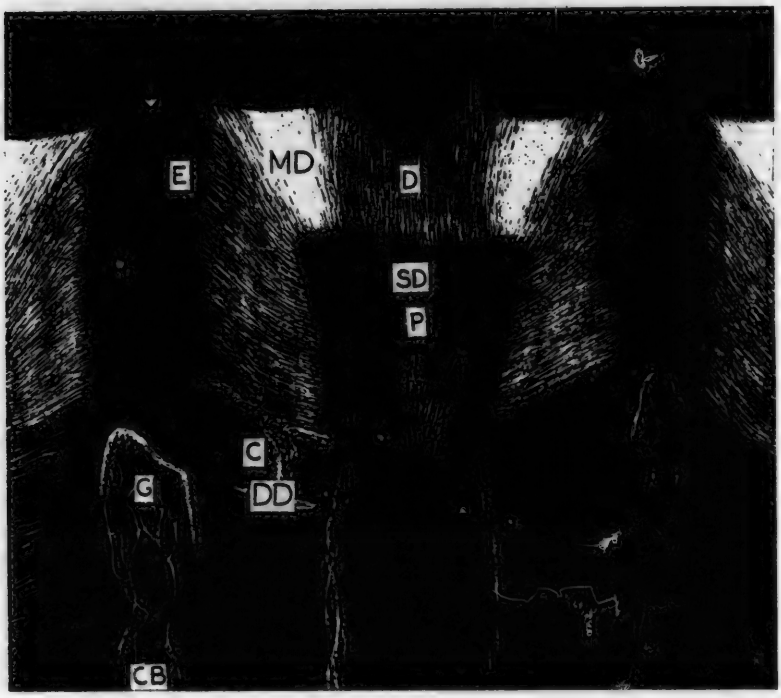


FIG. 3. ACUTE DENTAL ROOT CARIES

The crowns of teeth of old persons often are immune to dental caries; they have become highly impermeable due to changes in the dentinal tubules and the formation of secondary dentin (SD) in the pulp chamber (P). The neck of the tooth frequently becomes denuded, exposing this, more permeable area to acute dental root caries (C). The layer of decalcified dentin (DD) is fairly thick and the penetration of the lesion more rapid than that of chronic dental caries. The vascularity of the pulp is low. ARROW = entrance for food impaction, forcing the gingival papilla (G) away from the cervix of the tooth. CB = crest of the alveolar process.

term chronic as applied to this classification of caries, does not mean that at one time the lesion was acute, followed by a second stage of reduced severity. Chronic dental caries starts in the very beginning as a slow destructive process. The form of the lesion (*fig. 2*) is different from that of the acute

crown type; it has a larger entrance, even in its incipiency which precludes the undisturbed retention of food, hence fermentation is reduced; the saliva also, can more readily retard acid formation in this type of lesion. The layer of decalcified dentin (*DD*) is thin, covering the hard, still calcified tissue. The tubules in this area have been affected gravely throughout their entire length and the pulp has had an opportunity of forming secondary dentin, thus sealing off many of the channels leading to the lesion. When this destructive process affects the approximal surfaces, the contact points are often lost, so that the patient becomes uncomfortably conscious of food impaction. This may result in cervical caries (*fig. 2, CC*) and in gingival disturbances.

(3) *Acute dental root caries* usually affects the teeth of persons past middle age. The crowns of such teeth are often either completely non-carious, or at most have small occlusal fillings. These lesions affect the roots of the teeth, usually at the interproximal surfaces; food impaction seems often to be the external cause. *Figure 3* shows the form of the lesion in diagram and indicates the rapidity of its penetration towards the pulp. The contact points of two neighboring teeth often are lost by abrasion, allowing food impaction; this forces the gingiva from the cervix of the tooth and a cavity forms on the cementum. The rapidity of its penetration varies, it seems to be greater than that of the chronic type and somewhat slower than the acute crown lesion of dental caries.

A previous classification of dental caries was set up (2). In this a detailed description of the lesions was given, but it was impossible to publish adequate illustrative material. The lesions were described originally as acute, chronic and senile. Since the publication of this article, it was realized that an improvement could be made in these terms. Edmund Applebaum has suggested a modification which seems to define the lesions more clearly. The term acute root caries particularly is more pertinent, inasmuch as it localizes the lesion definitely. It also obviates the mention of the word senile, which few patients like to hear as applied to their condition.

## FACTORS AFFECTING THE RATE OF PENETRATION OF DENTAL CARIES

There seem to be at least three factors which determine the rapidity of penetration of the lesion of dental caries. They are (1) systemic disturbances (2) character and location of the lesion, (3) change in permeability of the teeth.

(1) The discussion of the influence of systemic conditions on the welfare of the teeth does not lie in the scope of this paper. Little, as yet, is known except that observations on children and animals indicate that the calcium-phosphorus-vitamin D complex is actively concerned in maintaining sound teeth.

(2) The differences in the forms of the lesions of acute and chronic dental caries seem to have a marked effect on the rapidity with which teeth are destroyed. The small entrance of the former prevents the saliva from quickly diluting the acid produced in the cavity by copious food fermentation. As a result of this probable higher acid concentration, the destructive process is more active and the lesions of acute crown and root caries penetrate more rapidly towards the pulp. The chronic lesion of dental caries, on the other hand, has been shown to have a wide entrance, thus allowing less opportunity for food retention and a more ready washing-out of the cavity by the saliva. Hence one reason for the slow penetration of the chronic lesion of dental caries seems to be because the amount of acid probably is less in this type of lesion than that formed in the bottle-shaped lesion of acute caries.

(3) Reduced permeability of the teeth was first defined by Pickerill (3) and emphasized by Beust (4). This factor appears to operate ten to fifteen years after the eruption of each tooth in civilized people (according to Pickerill, it occurs earlier in primitive people). Beust stressed particularly the difference in permeability in the teeth of young and old individuals. *Figure 1*, for instance, shows a tooth of a young person in which the dentin appears translucent, indicating that the tubules are open; this tooth was highly permeable. *Figure 3*, on the other hand, shows the more impermeable tooth of an old person, in which the dentin is fairly opaque, indicating that many of the tubules are closed. The manner in which the permeability of the dentin and the enamel is reduced is as yet in doubt. Suffice it to say that the permeability is reduced, as has been shown in different ways by Beust (5), Bödecker and Applebaum (6), Hodge (7), Karlström (8) and others.

Before discussing the probable resistance of the teeth against caries, created by a reduced permeability, it is necessary to examine the physical differences of the dental tissues between recently and long erupted teeth. The dentin and enamel of a newly erupted tooth do

not seem to have their full quota of mineral salts, as shown by Karlström (8), Hodge (7) and others, causing them to be in a comparatively permeable condition. In this state, the external attacking factor of dental caries (acid) can readily penetrate the enamel and dentin, *if* the natural protection of the teeth (saliva and dental lymph) is low. In this case the result during youth is the development of the rapidly penetrating, acute lesion of dental caries.

After ten to fifteen years (the time varies in different persons) the post-eruptive calcification of the *crown* is more advanced, resulting in a marked reduction of its permeability. If caries is active at this time, the lesions can penetrate but slowly; chronic dental caries is the result. Frequently, even in older persons, the *root* retains its permeability, due probably to the absence of secondary dentin. Should the gingival tissue recede under these conditions, and should the natural protection of the teeth be low, acute root caries may develop, often leaving the less permeable crown of the tooth immune to attack.

## AGE DISTRIBUTION

The commonly observed age distribution of the three types of dental caries is as follows: the acute crown type is usual in children and young adults; the chronic lesions in middle age; and the acute root type in the later years of life. It should be borne in mind that although this is the usual occurrence, there are frequent exceptions. Adolescent persons, for instance, who are only slightly susceptible to dental caries may have the chronic type in early adult life. After these defects have been repaired, there may be no recurrence till late in life, when they may become susceptible to acute root caries. Another exception has been noted in some middle aged persons who have gone through life with but little tooth decay, until, following some severe systemic disturbance, caries become active.

## SUMMARY

The clinically observed variations in the activity of dental caries seem to be related to these three factors:

(1) *Systemic conditions.* A disturbance of some phase of the calcium-phosphorus-vitamin D complex appears to increase the activity of dental caries.

(2) *Character and location of the lesions.* The lesions of dental caries can be divided into three groups: (a) acute dental crown caries, (b) chronic dental caries, and (c) acute dental root caries according to their form, location on the teeth and their different activity.

(3) *Change in the permeability of the teeth.* The comparatively high permeability of recently erupted teeth allows the external destructive force (acid) to penetrate rapidly, resulting in *acute* dental caries. On the other hand, the lowered permeability of matured teeth is a barrier to this attacking force; hence the slower penetration of the *chronic* type of the lesion.

The fact that there exists a variation in the activity of acute dental crown caries and chronic dental caries, suggests that the teeth offer a varying resistance to external attack.

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# INHERITED CONGENITAL ABSENCE OF SIX INCISORS, DECIDUOUS AND PERMANENT[1]

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Congenital absence of teeth is not of general occurrence, nor is it extremely rare. Much discussion has been advanced in recent years as to the etiology of the abnormality. Endocrine glands, syphilis, diet and heredity all have been discussed in this connection, and no doubt all are correct to a certain extent. It appears, however, that the hereditary factor is responsible for a large percentage of these conditions. Since early in the nineteenth century reports have appeared tracing dental anomalies through several generations. Some of these family histories have been dependable; others, unfortunately, have not.

A case was referred to the author following through at least four generations of the patient's family. There were forty-six members, eighteen of whom had at least six anterior teeth missing congenitally in both dentitions. (Chart I.)

The patient (D 18), a twin, was a white male twelve years of age, of average height and weight, intelligent and in good health. Examinations by a pediatrician and endocrinologist were entirely negative. Wassermann and Kahn tests were negative.

A clinical examination of the child revealed the following teeth to be caries free and firmly fixed in the jaws:

$$\text{R.}\ \frac{6,V,IV,III,1}{6,V,IV,III}\ \Big|\ \frac{1,III,IV,V,6}{III,IV,V,6}\ \text{L.}$$

The occlusion of the posterior teeth was normal and the gingivae were healthy. The teeth showed no defects, and no calcular deposits were found.

[1] Presented at the Fourteenth General Meeting of the International Association for Dental Research, Louisville, Ky., March 14–15, 1936; *J. Den. Res.*, 15: 311, 1936.

Roentgenological examination (Fig. 1) revealed the complete absence of the following permanent teeth:

$$\text{R. } \frac{2}{5,2,1} \Big| \frac{2}{1,2} \text{ L.}$$

The left mandibular second bicuspid was malposed, growing in a horizontal position with the occlusal surface facing distally. This is

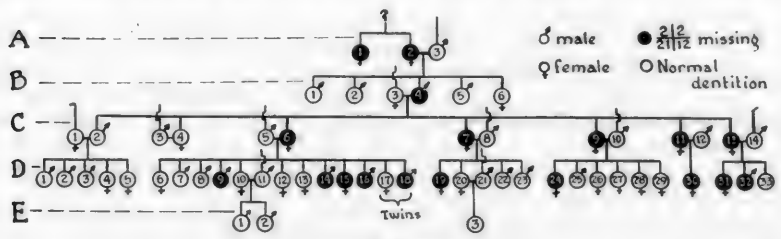


CHART I. Genealogy

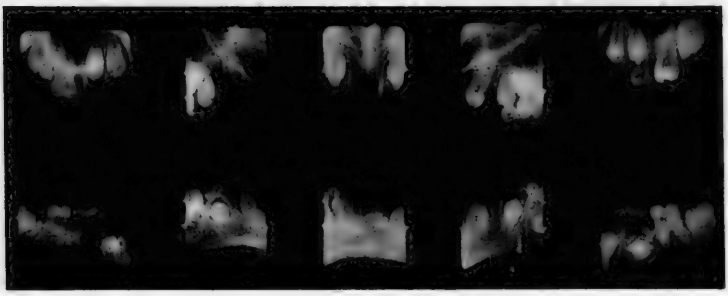

FIGURE 1. Intra-oral x-rays of D 18

particularly interesting when compared with the right side where the corresponding tooth is missing. The twin of the patient (D 17), was a female of larger stature, in good health, and with normal, good dentition.

The mother of the patient (C 6) had had several extractions previous to our examination of her teeth. The teeth present were:

$$\text{R. } \frac{8,7,5,4,3,1}{8,7,4,3} \Big| \frac{1,3,4,5,7,8}{3,4,5,7,8} \text{ L.}$$

Several teeth admittedly had been extracted, but the mother insisted, under close questioning, that R. $\frac{2}{2,1}\Big|\frac{2}{1,2}$ L. had never been present either in the permanent or deciduous dentitions. A root fragment had been retained from the mandibular left first molar.

X-ray examination of one of the patient's sisters (D 15) revealed the following teeth absent: R. $\frac{6,4,2}{2,1}\Big|\frac{2,4}{1,2}$ L. $\underline{\frac{6,4}{}}\Big|\underline{\frac{4}{}}$ had been extracted. Here, also, the incisors had never been present in the deciduous or permanent dentitions. The difference between the sister's and brother's teeth is found in the mandibular second bicuspids. In the girl, the bicuspids are present and normal. The deciduous cuspids have been retained and have shifted to the position ordinarily occupied by the lateral incisors.

Inquiring into the history of the family brought out the facts upon which the genealogical chart (Chart I) was based. It may be assumed that the chart is accurate, inasmuch as the statements about the members of the family were substantiated by similar statements by relatives in other parts of the country. Also, the appearance of the face and teeth is so characteristic where the anomaly occurs, that there is no difficulty, even to the untrained eye, in identifying it.

In studying over the chart it may be seen that thus far the anomaly has not been transmitted through normal individuals (C 2, D 10, and D 20). Also it is to be noted that affected females (A 2, C 6, C 7, C 9, C 11 and C 13) have had four types of offspring, namely: normal females, affected females, normal males, and affected males. At the present time only one affected male (B 4) has had any children. Of the seven children, he had one normal male, five affected females, and one normal female. As to the etiology, little can be said except that mutations appear from time to time from no apparent cause and the new characteristics are transmitted, modified, or lost depending on various factors, including the types of mating.

Assuming that a sex-linkage existed it would have to be a linkage on the "X" chromosome and the anomaly would appear when just one chromosome was affected. (Chart II.)

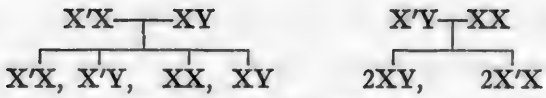


CHART II. "X" chromosome linkage

Hence, normal males and females would not transmit the defect, affected males would transmit it to all the female progeny, but not to the male. This plan fits perfectly to the family described except for C 4, the only unaffected female of B 4.

If future offspring fail to demonstrate a maintenance of the possible sex-linkage, the instance will at least be recognized as an example of a simple Mendelian dominant.

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# THE RELATION BETWEEN THE SHAPE OF THE OCCLUSAL SURFACES OF MOLARS AND THE PREVALENCE OF DECAY. II[1]

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In a previous paper (1) data were presented on the correlation of frequency of caries to the angles of inclined planes of cusps of the upper right first *permanent* molar. This report deals with an extension of the study to include measurements on the upper right second *deciduous* molar. The procedure was essentially the same as before. Samples were selected, the index of steepness of the occlusal valley measured, and the data tabulated and evaluated with reference to presence or absence of caries in the central pit area. The mass from which the sample was taken is the totality of children of New York City; of these the Children's Clinic of New York University Dental College has on file records and models of about 300 children. It is assumed that the manner in which this clinic selected its patients resulted in no statistical pre-selection correlated in any way with the condition of the upper right second deciduous molar.

The cases included children between 2 and 8 years of age (average age, 4). Measurements, accurate to 0.1 mm., were made as before from stone models with a Stanton Surveyor. Of the 300 specimens, 50 were non-carious in the central pit area and the remainder carious in this area. The 50 non-carious cases and 50 carious cases were measured in the manner previously described. Teeth with fillings or badly broken structure were not chosen; otherwise the selection of carious teeth was at random. Caries in the central pit area were recorded when present. Measurements were made from the tip of the mesio-lingual cusp to the tip of the mesio-buccal cusp (width), and from the tip of the mesio-lingual cusp to the deepest point of its

[1] Read at a meeting of the New York Section, International Association for Dental Research, February, 1935.

TABLE 1

*Upper right second deciduous molars*

| "r" | WITH CARIES | WITHOUT CARIES | TOTAL | WITH CARIES (ADJUSTED) | WITHOUT CARIES | TOTAL (ADJUSTED) |
|---|---|---|---|---|---|---|
| .31 | | 1 | 1 | | 1 | 1 |
| .32 | | 1 | 1 | | 1 | 1 |
| 3 | | 1 | 1 | | 1 | 1 |
| 4 | | 2 | 2 | | 2 | 2 |
| 5 | | 1 | 1 | | 1 | 1 |
| 6 | | | | | | |
| 7 | | 3 | 3 | | 3 | 3 |
| 8 | | 3 | 3 | | 3 | 3 |
| 9 | | 1 | 1 | | 1 | 1 |
| .40 | 1 | 6 | 7 | 5 | 6 | 11 |
| 1 | 1 | | 1 | 5 | | 5 |
| 2 | 2 | 3 | 5 | 10 | 3 | 13 |
| 3 | 4 | 2 | 6 | 20 | 2 | 22 |
| 4 | 5 | 3 | 8 | 25 | 3 | 28 |
| 5 | 2 | 4 | 6 | 10 | 4 | 14 |
| 6 | 1 | 3 | 4 | 5 | 3 | 8 |
| 7 | 2 | 3 | 5 | 10 | 3 | 13 |
| 8 | 1 | 4 | 5 | 5 | 4 | 9 |
| 9 | 1 | 1 | 2 | 5 | 1 | 6 |
| .50 | 5 | 2 | 7 | 25 $\frac{7.4}{17.6}$ | 2 $\frac{.6}{1.4}$ | 27 $\frac{8}{19}$ |
| 1 | 2 | 3 | 5 | 10 | 3 | 13 |
| 2 | 4 | | 4 | 20 | | 20 |
| 3 | | | | | | |
| 4 | 3 | 1 | 4 | 15 | 1 | 16 |
| 5 | 3 | 1 | 4 | 15 | 1 | 16 |
| 6 | | | | | | |
| 7 | 2 | | 2 | 10 | | 10 |
| 8 | 2 | 1 | 3 | 10 | 1 | 11 |
| 9 | | | | | | |
| .60 | 2 | | 2 | 10 | | 10 |
| 1 | | | | | | |
| 2 | 1 | | 1 | 5 | | 5 |
| 3 | 1 | | 1 | 5 | | 5 |
| 4 | | | | | | |
| 5 | 1 | | 1 | 5 | | 5 |
| 6 | 1 | | 1 | 5 | | 5 |
| 7 | 1 | | 1 | 5 | | 5 |
| 8 | | | | | | |
| 9 | | | | | | |
| .70 | 2 | | 2 | 10 | | 10 |
| | 50 | 50 | 100 | 250 | 50 | 300 |

inner outline (depth). The index of steepness,[2] r = width/depth, ranges from 0.31 to 0.70.

Table 1 shows, on the left, the distribution of the 50 non-carious and 50 carious teeth against r; it is obvious that the indices of steepness for the carious teeth are distributed over a range of larger values than those for the non-carious teeth. The cases collected under the rubic r = 0.50 are those with r between 0.495 and 0.505. Since the original sample included 300 teeth of which 250 were carious, the measurements on the 50 carious teeth may be adjusted by multiplying the number of occurrences of each r by 5 so that the occurrences of values for carious teeth total 250. Then, the

TABLE 2

*Distribution of carious and non-carious teeth in groups 1 and 2*

| | CARIOUS | NON-CARIOUS | TOTAL |
|---|---|---|---|
| | % | % | % |
| Group 1<br>Shallow fissures<br>$r \leqq 0.50$ | 36 | 14 | 50 |
| Group 2<br>Deep fissures<br>$r \geqq 0.50$ | 47 | 3 | 50 |
| Total..... | 83 | 17 | 100 |

values so obtained for 250 carious teeth are added to the values measured on the 50 non-carious teeth to give a total sample of 300. This process involves two assumptions, (1) that the distribution of r's in the 50 carious teeth measured is the same as in the remaining 200 teeth, (2) that the distribution of carious vs. non-carious teeth in the sample of 300 is typical of this distribution in the mouths of all New York children. On the right of Table 1, the adjusted values are given. The column of adjusted totals was used

[2] As noted in the diagram given in the previous paper (Fig. 1, p. 126) the perpendicular height of the mesio-buccal cusp XY = A (depth), and the horizontal distance between the two cusps ZM = B (width), were the dimensions recorded; and A/B = R (index of steepness) was the variable of statistical distribution. The corresponding quantity for deciduous teeth will be called r, to distinguish it from the index for permanent teeth.

to determine the point at which the cases could be divided into two equal groups. This point falls at r = 0.50, the 27 cases having this value being divided so as to yield two equal groups, 8 being added to the group r <0.50, 19 to the group r >0.50. The other adjusted values for r = 0.50 were divided proportionately, as indicated by the fraction to the right of the respective columns. The distribution of

FIG. 1. Indicates roughly appearance of mean angle in each of steepness groups.

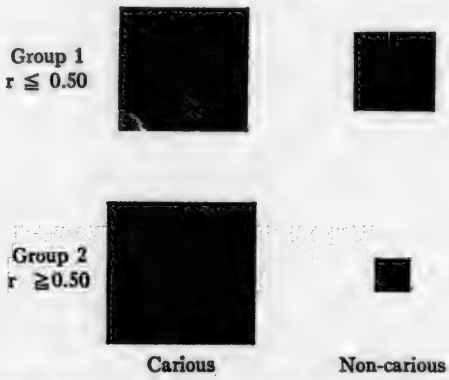


FIG. 2. Distribution diagram, to show, pictorially, relative magnitudes of distribution given numerically in Table 2. Area of each square proportional to relative frequency with which cases picked at random fall into that particular classification. Squares lie in same relative positions as corresponding numbers in Table 2.

carious and non-carious teeth between the two groups is given in percentages of the total number of teeth in Table 2.

*Fig. 1* indicates roughly the appearance of the mean angle in each of the steepness groups. *Fig. 2* is a distribution diagram to show the relative magnitudes of the distributions given numerically in Table 2. The area of each of these squares is proportional to the relative frequency with which cases picked at random should fall into that particular classification. These squares lie in the same relative posi-

tions as their corresponding numbers in Table 2. From this figure it appears that (a) if an upper right second deciduous molar is known to be carious, it is more likely to belong to group 2 than to group 1; (b) if it is known to be non-carious, it is more likely to belong to group 1 than to group 2; and (c) if it belongs to group 1, it is less likely to be carious than if it belongs to group 2. Such a deciduous molar picked at random, however, is much more likely to be carious than not; *i.e.*, 83 per cent are carious. If by some means (2) the slopes of all deciduous teeth were reduced, before decay, so that all fell into group 1, the probability of caries would be reduced to 72 per cent.

Further studies are in progress.

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# DENTAL LESIONS IN RELATION TO PULMONARY TUBERCULOSIS

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The dental staff of the Montefiore Hospital has undertaken an intensive investigation to determine whether there are significant oral signs of tuberculosis. In many of the reports available, conclusions were based on a small number of cases and inadequate case histories and clinical criteria. During the first two years of this study, 1500 patients in all stages of tuberculosis were studied to determine whether dental and periodontal signs could be established as pathognomonic of tuberculosis.

A committee, composed of members of the dental staff, appointed to study the subject devised a special chart to record the clinical findings of the investigators. The literature was reviewed and the research group then systematically gathered and compiled the clinical data which is the basis for this preliminary report.

Opinions regarding the teeth and supporting structures in the tuberculous, are quite divergent. Obviously only an intensive, carefully planned investigation with ample and varied clinical material could shed additional light on the subject. Members of the staff were impressed by the normal appearance of the teeth and their supporting tissues and the absence of unusual signs in the mouths of tuberculous patients on whom they had been working since 1921.

As the work progressed, it seemed that the results would confirm the earlier experiences of the staff, *i.e.*, that there are no specific oral signs pathognomonic of tuberculosis. The data have been analyzed and compiled in a statistical table. (Table 1.)

The preliminary results of our studies indicate the following:

(1) There is no more dental or periodontal disease present in tuberculous individuals than in healthy persons.

TABLE 1

*Statistical report on patients*

M.—male, F.—female, G.—generalized, L.—local, S.—slight, M.—moderate, MA.—marked.

| AGE | NO. | SEX | | MARITAL STATUS | | LESIONS ON TEETH | | | | | EDENTULOUS | MOBILITY | | | GINGIVAL MARGIN | | | | | | LESIONS ON TONGUE[1] |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | M. | F. | Single | Married | Hypoplasia | Erosion | | Abrasion | Attrition | | S. | M. | MA. | Recession | | Pain | Gingivitis | Bleed. | Supp. | |
| | | | | | | | G. | L. | | | | | | | G. | L. | | | | | |
| 15–20 | 108 | 64 | 44 | 108 | | 12 | 4 | 4 | 4 | 4 | | 4 | | | 24 | 12 | | 44 | 32 | 16 | |
| 21–25 | 280 | 152 | 128 | 220 | 60 | 32 | 24 | 20 | 24 | 32 | | 24 | 4 | | 52 | 20 | 12 | 124 | 88 | 24 | |
| 26–30 | 288 | 148 | 140 | 168 | 120 | 24 | 24 | 44 | 28 | 20 | 4 | 12 | 12 | | 80 | 60 | 8 | 160 | 108 | 20 | |
| 31–35 | 212 | 80 | 132 | 60 | 152 | | 24 | 32 | 20 | 48 | 8 | 20 | 16 | 4 | 88 | 40 | 8 | 128 | 72 | 56 | |
| 36–40 | 216 | 104 | 112 | 48 | 168 | 8 | 24 | 20 | 36 | 48 | 20 | 44 | 16 | | 80 | 32 | 8 | 116 | 52 | 36 | 4 |
| 41–45 | 132 | 104 | 28 | 24 | 108 | 4 | 4 | 4 | 16 | 28 | 24 | 16 | 16 | 4 | 52 | 4 | | 56 | 20 | 16 | |
| 46–50 | 136 | 80 | 56 | | 136 | | 8 | 8 | 20 | 44 | 36 | 4 | 24 | 4 | 44 | 20 | | 64 | 16 | 20 | |
| 51–55 | 136 | 100 | 36 | | 136 | | 12 | 12 | 24 | 40 | 28 | 16 | 16 | | 60 | | | 36 | 24 | 28 | |
| 56–60 | 68 | 56 | 12 | | 68 | 12 | | 4 | 12 | 16 | 24 | 4 | 12 | | 28 | 12 | 8 | 16 | 8 | 20 | |
| 61–65 | 48 | 20 | 28 | 2 | 46 | | | | 4 | 4 | 36 | | | | | | 4 | 8 | | | |
| 66–70 | 20 | 16 | 4 | | 20 | | | | 4 | 8 | 12 | | | | 4 | | | 4 | | | |
| 71–75+ | 8 | 4 | 4 | | 8 | | | | | | 6 | | | | | | | | | | |
| | 1652 | 928 | 724 | 630 | 1022 | 92 | 124 | 144 | 192 | 276 | 198 | 144 | 116 | 12 | 512 | 200 | 48 | 756 | 420 | 236 | 4 |

[1] No lesions on mucous membrane.

(2) Gingivitis and erosions, although present in some cases, are not found in greater abundance in tuberculous than in healthy individuals. Even in the presence of extreme debilitation in patients suffering from far advanced pulmonary tuberculosis, there is no unusual increase in the incidence of caries, erosions and alveolar destruction.

(3) A rather unusual observation was made in cases of numerous patients in whom caries and erosions were traced to the use of lozenges with high sugar content, a condition described by Miller (1). These patients, on initial examination revealed no unusual dental or periodontal disorders; however, several months later, they were found to be suffering with carious teeth. It was discovered that these patients were using lozenges to relieve the dryness of their mouths and throats and to prevent excessive coughing. This may explain to a degree why caries and erosions have been associated with tuberculous patients.

(4) Without resorting to laboratory procedures and depending entirely on clinical observations, it seemed that there were no sequelae of avitaminosis or metabolic disorders. Fishberg (2) stated, "Many authorities, notably Labbe and Gollipe, Steinitz and Weigert, have found no demineralization in tuberculosis." Wells, De Witt and Long (3) summarize the problem by saying, "Despite the widespread belief that tuberculosis is characterized by a marked loss of inorganic elements, there is no evidence for such a process in carefully controlled observations on mineral metabolism." In the vast majority of tuberculous patients, the basal metabolic rate is quite normal in the absence of high fever and it may be stated that as yet, no metabolic disturbance has been shown to precede constantly, or even often, the onset of pulmonary tuberculosis. It would be a serious error to employ the basal metabolic rate as a diagnostic sign in tuberculosis. There was no evidence of decalcification in our cases that could be definitely the result of a demineralization process initiated by tuberculosis.

(5) X-ray examinations of the teeth and their supporting structures revealed no unusual changes which could be associated with tuberculosis.

(6) Secondary lesions, occurring as a rule late in the course of pulmonary tuberculosis, in the form of ulcers on the tongue or buccal membranes, were uncommon. These lesions were too infrequent to be of value as diagnostic aid.

(7) Age and sex play no rôle in the incidence of lesions involving the teeth and supporting dental structures.

(8) Neglect of the mouths of tuberculous patients has been responsible to a marked degree for the development of dental symptoms. Since this is

equally true for normal individuals, the dental disorders in the tuberculous bear no relationship directly to the tuberculous process.

(9) Vincent's infection does not occur more frequently in tuberculous patients than in other individuals.

Our studies have convinced us that there are *no pathognomonic dental or periodontal signs of pulmonary tuberculosis* in the mouths of tuberculous patients.

From the statistical chart (Table 1) it is interesting to note that although 1652 cases were examined, only 4 cases had lesions on the tongue, 756 cases or 46% had gingivitis, 420—bleeding gums, and 236—suppuration, 512—generalized recession, 200—localized recession. The 756 cases of gingivitis are no different clinically than gingivitis in patients of normal health; there being no associated symptoms which would be pathognomic of tuberculosis. The same applies to the other conditions studied.

The clinical observations are now being checked by laboratory studies.

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